The New England
Journal of Medicine

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Volume 247 SEPTEMBER 25, 1952 Number 13

SURGICAL CLOSURE OF DEFECTS OF THE INTERAURICULAR SEPTUM BY USE OF AN ATRIAL WELL*

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BOSTON

DEFECTS of the interauricular septum in the human being carry a variable prognosis. Small openings are known to be tolerated rather well through a long span of life. Large defects give left-to-right shunts that enormously increase the flow of blood through the right side of the heart and the pulmonary circuit, thereby leading to right-sided heart failure in early or middle ages. The gravity of the problem makes it quite worth while to develop methods whereby defects of moderate or large size can be closed surgically. This communication summarizes certain of our investigations, which were made in the effort to reach this goal.

Various methods have been suggested or have actually been used in the attempts to close atrial septal defects. Those [1, 2] who have been developing extracorporeal pump-oxygenators hope to divert blood from the heart long enough to permit opening of the organ and closing of a hole in the septum under direct vision. Efforts have been made to use such an apparatus to permit closure of a septal defect in man[1] but as yet no successes have been reported. Murray[3] has passed mattress sutures of silk or strips of fascia through the heart, attempting to place these in the plane of the septum. These can partially block a septal opening, and if the mattress stitches are drawn close together the anterior and posterior walls of the auricles can be brought closer to one another, thereby partially reducing the size of the hole in the septum. The method can hardly be expected to give complete closure of a septal opening. Swan et al.[4] have inverted the right and left auricular appendages to the septum so that a septal opening can be plugged by holding the inverted appendages in this coapted position with mattress stitches passed through them. Such inversion was apt to block pulmonary veins, and the method has been abandoned. Cohn[5] suggested pushing in the right auricular wall to plug a septal opening, suturing the inverted wall to the septum (around the periphery of the defect). No report of the successful use of this method has been published. Bailey[6] has employed a modification of Cohn's technic, by inserting a finger through the auricular appendage into the auricle so that the digit can guide the placement of the sutures used for drawing and attaching the lateral auricular wall to the septum at the periphery of the defect, thereby plugging the hole. This method might be of great value for the treatment of an opening that lies in the middle portion of the septum, but it is impractical for treatment of the vast majority of defects since they lie high, low or forward in the septum. To close such openings by inversion of the auricular wall would almost certainly obstruct the orifice of the superior vena cava, the inferior vena cava or the tricuspid valve. Hufnagel and Gillespie[7] have devised pairs of flat, plastic buttons, which can be screwed together, one disk to be placed on the left side of the septum while the other rests on the right of the septum, covering any hole in it. These buttons, put into place by a simple and ingenious handle, have been found to be effective in closing septal defects created in dogs. They will probably be of little value for human patients, in whom the septal defects seldom have a rim around the entire periphery; it is essential to have a complete rim to hold the double button in place.

Each of the methods enumerated above for closing a defect of an auricular septum has serious technical disadvantages, and we do not believe any of them will have more than a limited usefulness. Pursuing a somewhat different line of thought, we considered it possible to attach temporarily some sort of cone-shaped rubber bag (open at the top) to the right auricular wall so that the auricle could be opened and blood allowed to rise up into the rubber "well." Through this column of blood, it might be possible to pass the operator's fingers down into the auricle, to find the septal opening, and to

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close it either by direct suture or by the onlay of a piece of flat substance (such as a plaque of fascia, pericardium or plastic). Investigations were made in the Laboratory for Surgical Research at the Children's Hospital to determine, first, how it might be possible to attach such an appliance to an auricle and, secondly, how the heart might stand opening of its auricle to atmospheric pressure. This report is a summary of our findings.

Laboratory Studies

With mongrel dogs, ranging in weight from 17 to 26 kg., a study was made of various methods that would allow temporary attachment of a conical

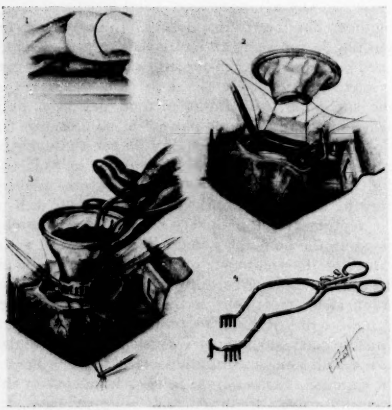

Figure 1. *Application of a Rubber Well to the Right Auricle. 1 = Position of animal for exposure of the right side of the heart. 2 = Pericardium opened, with a special clamp placed across the base of the atrium and the atrial wall slit open beyond this clamp; the rubber well is being sewed to the edges of the auricle with silk sutures. 3 = Position of silk sutures around the base of the well (the atrial clamp has been removed, and blood has been allowed to rise up into the well). 4 = Type of self-retaining retractor that is inserted into the well to spread open the orifice of the auricular wall.*

piece of pliable cellophane or rubber to an auricular wall in such a way that the seal would not leak blood and also in such a way that the auricular wall would not be left damaged after removal of the well. At first, it seemed that it might be possible to glue a "well" to the auricular wall with rubber cement, plastic cement or other adhesive substance, but all these were found to be unreliable. Later, it was determined that a well and the auricular wall could be held together satisfactorily with interrupted sutures of No. 0000 Deknatel silk. The completion of the union required no more than ten or fifteen minutes, and the junction could be made watertight.

After determining that it was feasible to attach some sort of bag or well to an auricle in a reliable way, we turned our attention to the careful designing of a rubber well that might best serve the purposes for which it was intended. After several different models had been constructed and tried a type that fulfilled all the requirements was arrived at (Fig. 1). It is a truncated cone, open at the top and bottom, the wall being made of pliable, but extremely tough, rubber 0.38 mm. in thickness. The lower rim, through which the anchoring silk stitches are passed, is reinforced by a double thickness of such rubber. The upper edge of the well incorporates a coiled spring, which expands and keeps open the mouth of the well. A well 10 cm. high was found to be adequate to contain the column of blood that rose in it when a dog's auricle was opened; the column seldom went above 5 or 6 cm.

After a well had been attached to an auricle, it was found desirable to have some sort of self-retaining retractor that could be inserted into the well and placed in such a way that the opening in the lateral wall of the auricle (and the attached ring of rubber) could be stretched open to make an orifice 3 or 4 cm. in diameter. After experiments with different forms of retractors, the instrument shown in Figure 1 was adopted.

A series of 114 dogs were used to develop proficiency in the technics of applying a well to an auricle. When a well was open and received blood from the auricle, clotting was prevented by the addition every few minutes of a few cubic centimeters of heparin solution; in no instance was there clotting in the well. During the time such wells were open to atmospheric pressure, observations were made on the cardiac action, and direct pressure readings were made with electrical manometers from the right auricle, the right ventricle and the carotid artery. Dogs, with the wells attached, were maintained under pentobarbital sodium (Nembutal) anesthesia, with positive-pressure ventilation of the lungs through an endotracheal tube. Detailed listing of these physiologic studies will be made elsewhere; general statements and conclusions regarding them are as follows:

Temporary clamping of an auricle, and placement of stitches in the auricular wall and septum, did not produce any disturbing irregularities of the heart; seldom were there more than a few dropped beats or extrasystoles.

The pulse rate rarely changed to any important degree.

The level to which blood rose in the well varied from 4 to 8 cm. above the lateral surface of the right auricle. This level varied a centimeter or two with the respiratory excursions.

At no time was air sucked down into an auricle.

The peripheral blood pressure was always at an adequate level.

The well could be left open for periods up to one hour (the longest period studied), and during such times the circulation could be maintained in a stable manner.

With an open well, one or several fingers could be passed down through the pool of blood so that the right auricle could be entered and its interior slowly and carefully explored by digital examination. It soon became evident that one could become quite proficient in recognizing the various landmarks such as the tricuspid valve, the coronary sinus, the eustachian valve, the orifices of both venae cavae and the various parts of the interauricular septum. Pieces of septum 1 to 3 cm. in diameter were then cut out so that the defects thus created could be closed by various methods and studies could be conducted of the comparative values of the several methods of septal closure. After these interauricular manipulations had been completed, the rubber well was removed, and the lateral wall of the auricle was closed with interrupted figure-of-eight stitches of silk.

Six general methods of closing interatrial septal defects were investigated. Detailed analysis of these will be published elsewhere, but the general features were so clear that they can be summarized briefly here.

Free grafts of pericardium could be sewed to the septum to cover over a defect, the graft being sutured to the septum with an appropriate number of interrupted silk sutures. These grafts were tolerated quite well, became incorporated into the substance of the septum and subsequently became smoothly covered by endothelium. Clotting on them was never a problem. The greatest drawback to the method was the fact that the patch of thin and soft pericardium was sometimes difficult to feel with the palpating finger in the auricle, thus complicating placement of the graft. Furthermore, the graft material, being soft and pliable, required a large number of sutures at its periphery to give complete septal closure.

Pieces of vein were turned inside out and used for free grafts to cover over septal defects. They had the same disadvantage noted for the pericardial grafts, and in addition they were inadequate when large openings had to be closed.

The tip of the right auricular appendage could be cut off and used as a patch to sew to the septum and thus to cover over the septal opening. This left an irregular wad of tissue attached to the septum, which, however, exhibited an amazing tendency to flatten down, become incorporated into the septum and smoothed over during the ensuing six to eight weeks. On the whole, the method was quite satisfactory, but it had the disadvantages of requiring the steps of amputating the auricular appendage to obtain the material to be used for the grafting.

Defects were closed by the use of Hufnagel prostheses, which consist of two button-like disks that can be screwed together by virtue of a threaded central stem that one of them carries. One of these plastic buttons could be placed on the left side of the septum, and the other on the right side. Provided there was a rim of tissue around the entire periphery of the septal opening, these buttons could be put in position within a few moments, completely blocking the opening, and were covered over by endothelium in a most astounding manner in a few weeks.

Defects were closed by the application over them of a sheet of plastic material, 0.5 to 1 mm. in thickness, cut to appropriate size and shape, which was sewed to the septum with interrupted silk sutures. Nylon and polyethylene were very good to work with. A piece could easily be cut out with scissors at the operating table; it could be pierced without difficulty by needles. The material had enough body and stiffness so that it could be readily felt with the palpating finger in the auricle. The material was tolerated extremely well, was seldom accompanied by any serious clotting and was covered over by endothelium quite rapidly, making a smooth interior for both auricles.

Some defects were closed by the use of interrupted silk sutures, the edges of the septum being grasped so that they could be drawn together. This method was not suitable for closure of large openings, because great tension on sutures made them tear out of the septum. For openings less than 1.0 cm. in maximum dimensions, such a closure by direct stitching was extremely satisfactory; the silks became incorporated within the septum and were covered over by a smooth endothelium, free of surface clot.

In summary, our various observations convinced us that septal defects that have margins of tissue around their entire periphery can be quickly and effectively closed by the use of Hufnagel buttons, that septal defects of almost any size, shape or position can be covered over effectively by the insertion and anchoring into place of a sheet of plastic material, and that defects smaller than 1.0 cm. can generally be closed by direct suture and approximation of the septal edges.

Clinical Application

After extensive trials with the use of auricular "wells" in experiments on animals, it was considered justifiable to attempt closure of defects of the interauricular septum in man. To date, 6 patients have been operated upon. For work on human beings, a rubber well has been constructed with a height of 15 cm., an upper-orifice diameter of 13 cm. and a lower-orifice diameter of 4 cm. For use with this, a larger self-retaining retractor has

been devised, its blades being of sufficient length to dip down into such a deep well. When the jaws are opened, the orifice in the lateral wall of the auricle (and in the base of the well) can be expanded to 6 cm. or more in diameter. A deep well was provided to take care of any excessively high column of blood that might be encountered in human patients. In the limited experience to date, it has not been necessary to use the entire height of the rubber bag at any time; the bag has generally been folded or rolled down upon itself so that only a third or a half of its height has been employed, thereby simplifying holding of the bag and working through it.

Six patients varying from four to sixteen years of age have been operated upon. The left-right flows of blood through these shunts were shown by preoperative catheterization to range from 6.7 to 22.3 liters per minute. Four of the children had been known to have some degree of right-sided heart failure and had been treated by digitalization, low-salt diet and other supportive measures.

In no case was there the slightest difficulty in attaching the rubber well to the enlarged right auricle. To sew the rubber appliance to the auricle required fifteen to a maximum of twenty minutes. In no operation was there leakage at the suture line. There has been no clotting within the well, the blood being kept fluid by regional heparinization. Examination of the peripheral blood after operation showed that the clotting time had returned to normal in each subject within four hours.

In the first case, the well was kept open for twelve minutes. This was tolerated very well, in spite of the fact that the child had been in heart failure prior to operation. With increasing experience with the operation on human beings, it became evident that there was little need for haste in closing the well. In 1 case, the well was open for two hours and five minutes. Therefore, one could be careful and deliberate about the intracardiac manipulations and repair.

Three methods of attempting septal closure have been employed. In the first 3 patients, defects varying from 2.5 to 5 cm. in maximum dimensions were found. Each had a rim of septum around approximately half the opening, the remainder of the orifice having no rim of tissue that could be grasped. Rather unwisely, double buttons of the Hufnagel type were put into place, in the hope that the grasping of a rim halfway around the button would anchor it securely enough to hold the appliance in place. Although it was realized that under such circumstances a button would not completely occlude the septal window, it was hoped that a baffle would be created that would greatly reduce the flow of blood through the shunt. All 3 of these cases ended disastrously, death occurring within a few days; in each, the button worked loose. In 2 cases it fell freely into the right or left auricle, and in the third it retained part of its septal attachment but turned over and partially obstructed the tricuspid valve. These bitter experiences make it quite evident that such buttons (held in place by merely screwing two of them together), although highly satisfactory in the animal experiment, in which there is a margin of tissue around the entire rim of the septal defect, are rarely safe in human beings, who are too likely to have incomplete rims around the opening and hence do not have sufficient substance to hold buttons in place securely.

In a fourth patient a large and high septal defect was found, measuring 4 cm. in diameter. This had a good rim of septum inferiorly but none superiorly. A sheet of polyethylene, 0.5 mm. thick, was used to cover the opening. It was cut into an oval 4.5 cm. wide and 5.0 cm. long, was securely anchored to the septal wall above and posteriorly, and was sutured to the septum inferiorly. Unfortunately, after the plastic was in place, it was found that it was too long; it covered the septal defect quite well but projected down unnecessarily over the annulus of the tricuspid valve. In spite of this obvious error it was decided to leave it, rather than to attempt replacing it with a smaller plaque. This patient was in reasonably satisfactory condition for three days and then suddenly died. At autopsy the septal defect was found to be completely closed off, but the excess plastic inferiorly was found to be underlain with clot, which had extended downward, partially obstructing the orifice of the tricuspid valve. The plastic plate had been too large and had not been anchored down sufficiently along its caudal border.

In the fifth case (Fig. 2), the septal opening was found to be 2 cm. wide and 3 cm. long. It was quite high. There was a good rim of septal tissue inferiorly and anteriorly, but none superiorly and posteriorly. A piece of nylon, 0.8 mm. thick, 2.5 cm. wide and 3.5 cm. long, was put in place to cover the septal defect. This was anchored inferiorly and anteriorly by interrupted stitches to the available rim of septum. Above and behind, the silk sutures grasping the edges of the nylon plate were run out through the auricular wall; as these sutures were drawn up and anchored externally, the plate could be snugly drawn up against the inner surfaces of the atrial wall. This patient, who was discharged from the hospital on the thirteenth day, is in excellent condition two months after operation and the cardiac murmur has disappeared. Postoperative catheter studies have not yet been performed.

In the sixth patient (Fig. 3), two septal openings were found. A centrally located one was 1.5 cm. long and 1 cm. wide. It was closed with interrupted sutures of silk. The second defect was larger and was situated posteriorly and inferiorly; it was 3.5 cm. long and a little more than 1 cm. wide. There was a good rim of septal tissue along the anterior and superior borders of the defect, but none below or behind. A series of mattress stitches were placed,

the available anterior margin of the opening being grasped, and the stitches being passed out through the posterior wall of the auricle and brought to the right of the right pulmonary veins. When these sutures were drawn snug and tied outside of the auricle, the septal edge was drawn posteriorly, and simultaneously the posterior wall of the auricle was drawn in forward. Both these movements combined

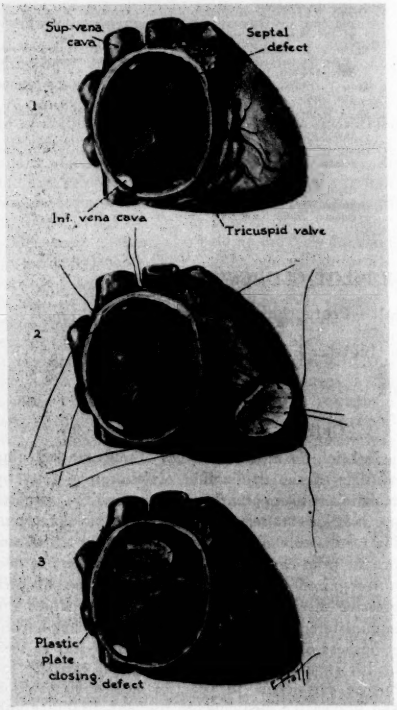

FIGURE 2. *Type of Repair Employed in Case 5.* 1 = *A very high septal defect, with a good rim of septal tissue inferiorly and anteriorly (no rim of septum above and posteriorly).* 2 = *Sheet of nylon of appropriate size and shape to cover the septal defect (silk sutures have been threaded through the rim of the septal defect inferiorly and anteriorly; superiorly and posteriorly silk sutures from the nylon sheet have been run out through the auricular wall.).* 3 = *Nylon sheet anchored in place by these various silk stitches.*

to close off the septal opening effectively and also to divert all blood from the right pulmonary veins into the left auricle. This patient made a rapid recovery, was discharged from the hospital in twelve days and was in excellent general condition two months later; the precordial murmur has disappeared.

From the observation on human beings to date, it seems clear that a rubber auricular well can be

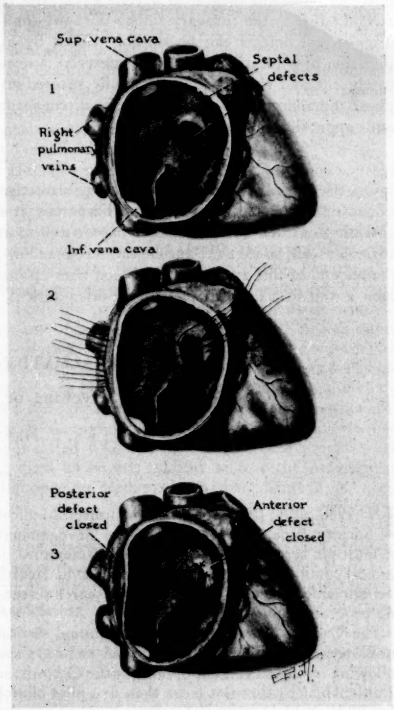

FIGURE 3. *Findings and Type of Surgical Therapy in Case 6.* 1 = *Two interauricular defects found. (The small anterior one had a good margin of tissue around its entire periphery; the posterior defect had a good rim of septum along its anterior margin, and no rim of tissue posteriorly.)* 2 = *Method of placing sutures for closure of the two defects. (The smaller, anterior opening is to be closed by approximation of its edges with interrupted silk stitches, and the larger, posterior defect treated by insertion of interrupted silk mattress stitches, grasping the margin of the septum and then running these stitches out through the posterior wall of the auricle, to the right of the pulmonary veins.)* 3 = *Sutures drawn tight to close both septal openings.*

attached to the human heart, that the heart can be opened into the base of such an appliance for a considerable time, and that the auricle can be entered through such a well to permit a defect of the interauricular septum to be closed. It is apparent that screw-on plastic buttons of the Hufnagel type will probably have only limited usefulness in human

patients because there is often an incomplete rim of septal tissue for the buttons to grasp. A plate of plastic material can be sewed and anchored into place to cover a septal defect, but it must not be made too large for fear of projecting into and giving obstruction of the orifice of a vena cava, the tricuspid valve or the coronary sinus. Without doubt, large septal defects can be suitably closed by application of a plastic sheet if the material is accurately cut and shaped and is carefully sutured into place. Smaller defects can be closed by direct suture and approximation of the septal edges.

Summary

On the basis of extensive laboratory investigations, a method has been developed whereby it is possible to attach an open rubber bag or well to an auricular wall, so that the auricle can be opened into the bottom of this attached sac. Blood rises up into such a well to a height equal to the intra-auricular pressure. Through the pool of blood, kept fluid by heparin, it is possible to enter the interior of the auricle, to locate accurately any septal defect and to close it in a careful manner. A large septal opening can be covered with an onlay sheet of plastic material, held in place by sutures. A small opening can be repaired by direct stitching and approximation of the septal margins.

References

1. Dennis, C., et al. Development of pump-oxygenator to replace heart and lungs; apparatus applicable to human patients, and application to one case. *Ann. Surg.* **134**:709, 1951.
2. Miller, B. J., Gibbon, J. H., Jr., and Gibbon, M. H. Recent advances in development of mechanical heart and lung apparatus. *Ann. Surg.* **134**:694, 1951.
3. Murray, G. Closure of defects in cardiac septa. *Ann. Surg.* **128**:843-853, 1948.
4. Swan, H., Maresh, G., Johnson, M. E., and Warner, G. Experimental creation and closure of auricular septal defects. *J. Thoracic Surg.* **20**:542-551, 1950.
5. Cohn, R., Experimental method for closure of interauricular septal defects in dogs. *Am. Heart J.* **33**:453-457, 1947.
6. Bailey, C. Material presented at a meeting of the College of Physicians, Cleveland, April, 1952.
7. Hufnagel, C. A., and Gillespie, J. F. Closure of interauricular septal defects. *Bull., Georgetown Univ. M. Center* **4**:137-139, 1951.

THE EVALUATION OF PILOT CLINICS*

The Mass Screening or Health-Protection Program

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BOSTON

IN 1948 and 1949 the Massachusetts Medical Society became interested in the development of multiple screening. After a series of meetings of several committees the Committee on Public Health recommended the establishment of a research screening program.

The Council of the Society, at its annual session in Worcester on May 23, 1949, voted to adopt the following motion: "It is the opinion of the Committee (Public Health) that not more than five pilot clinics should be held in pivotal localities throughout the State to offer, on a voluntary basis, health examinations under the auspices of district medical societies in co-operation with the community hospital and other interested groups."

A subcommittee on pilot clinics was appointed by the President and met on many occasions during 1949, at which time the committee passed on all the policies of the pilot clinic — determining what tests were to be used, how they were to be evaluated against the history and physical examination, and the contents of the letters that would be sent to the persons screened and to the doctors. More recent action of the Council has modified the composition of the Committee and made it advisory in function.

*Presented at the annual meeting of the Massachusetts Medical Society, Boston, May 20, 1952.

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Objectives of Multiple Screening

In brief, the modern concept of multiple screening consists of the application of laboratory tests by technicians to apparently well persons. The purposes are as follows: to refer persons with suspicious screening results to their family physicians for maximum benefit from early diagnosis and treatment; to find unrecognized or unsuspected early cases of progressive diseases for which testing procedures are administered: to induce people to develop a consciousness of the value of good health and to seek health supervision and early medical care; to influence physicians to practice health supervision and preventive medicine; to prevent disability and premature death from the development and progress of chronic illness; and to prevent economic loss to the individual and community from unemployment, prolonged medical care and welfare costs.

The ultimate objective of multiple screening is the motivation of people with suspicious results found in screening tests toward seeking early medical care; the incidental aim is to find unrecognized cases of progressive illness and also to obtain early treatment of these conditions in the physician's office. An indictment of multiple screening as a mass diagnostic procedure of little value is fallacious, since it is based on false premises.

Operation of Pilot Clinics

After a preparation of over six months the first pilot clinic was opened on December 1, 1949, at the New England Center Hospital and Joseph H. Pratt Diagnostic Hospital. It was sponsored jointly by the Massachusetts Medical Society and the State Department of Public Health. The program was designed to evaluate the specificity and sensitivity of the screening tests, to study the various motivations that brought people to the clinic, and to study the cost and administrative technics for the application of multiple screening by a health department, by a hospital for inpatients and outpatients, and by the private practitioner.

A research grant was obtained from the United States Public Health Service; and the Massachusetts Division of the Cancer Society and the Massachusetts Heart Association also made grants available for the operation of this clinic, at first through the Massachusetts Medical Society, later through the Department of Public Health.

The second session, also held at the New England Center and Joseph H. Pratt Diagnostic hospitals, began in September, 1950, and ended in June, 1951; during this period over 5000 persons were screened. During the interval a preliminary analysis of the records of the 2620 persons who had been screened at the first session was made. As a result of this study there were several significant changes in the procedures during the second session. These changes comprised both additions and omissions of tests and an acceleration of individual procedures. The physical examination was reduced from twenty minutes to ten minutes. It was believed that nearly all the positive signs being picked up by the multiple screening tests or by the self-screener could be evaluated in this ten-minute examination. The time per screening test was reduced from five to four minutes. The recording of temperature, pulse and respiration was omitted. Blood-sugar screening was being made by the use of the Clinitron, a newly developed instrument. An acetone determination was made on all specimens of urine in which the sugar level was higher than half of 1 per cent. Uterine cancers were screened by means of a beta-glucuronidase level on women in the mid-cycle between menstrual periods. Pelvic examinations with Papanicolaou smears were still made on all married women and, when indicated, on single women. During the latter part of the second session a modified electrocardiographic "tracer" with one lead was made on each person. For purposes of evaluation repeat cardiograms with multiple leads and repeat x-ray films, 14 by 17 inches, as well as a glucose tolerance test, were made. These confirmatory procedures were undertaken to check the validity of the abbreviated tests.

The third session of the pilot clinic was begun in March, 1952, at the Peter Bent Brigham Hospital and is still underway. Here the physical examination is being omitted, and for the first time a true multiple screening clinic is in operation.

The fourth session of the clinic started in Belmont, also in March, and was discontinued in May. It employed the following tests: self-screener or clinical history; blood test for serologic findings, sugar and hemoglobin; urine test for sugar and albumin, and acetone if indicated; vision test; hearing test; x-ray examination (film 4 by 5 inches); measurement of height and weight; recording of blood-pressure readings; hematest for occult blood; Papanicolaou smear on women over thirty-five or with symptoms; and one-lead electrocardiograms.

Held in a public-school building four times a week, with the approval and advice of the physicians of the town of Belmont, this clinic was conducted by technicians, with no physician present. Sponsored jointly by the Belmont Health Department and the Massachusetts Department of Public Health, this clinic received unusually good reception on the part of both the public and the doctors, as determined by an opinion poll. Of 22 general practitioners interviewed, 18 were very enthusiastic, 3 were somewhat critical, and 1 was doubtful. It has been our experience that as doctors know more of the clinic and have patients referred to them, their enthusiasm for the program grows. Physicians who are not acquainted with the operation of the program are inclined to be critical and to fear the program as the opening wedge of state medicine.

Pilot Clinics As Research

The Massachusetts Medical Society has endorsed mass screening as a case-finding method for tuberculosis, diabetes and cancer. The American Medical Association, together with the American Hospital Association, the American Public Health Association and the American Public Welfare Association, sponsored, through the Commission on Chronic Illness, a conference on the Prevention of Chronic Diseases held in Chicago, on March 12–14, 1951.

The Commission encouraged the evaluation of screening procedures and the development of a center equipped to test and develop mass screening procedures with special reference to follow-up study. The pilot clinics, as jointly sponsored by the Massachusetts Medical Society and the Department of Public Health, have been carrying on the very program that the American Medical Association and the other co-sponsors of the Conference on the Prevention of Chronic Diseases recommended in their published report. Thus, the pilot clinic has served as a pattern for the development of evaluation and research studies of mass screening technics. Since it has proved that a reasonable amount of research data will have been obtained in the near future, the pilot clinics will be discontinued at that time.

Evaluation

Each person was sent, prior to his visit, a 222-question self-screener or history. This history was completed at the clinic, after which that person was put through the various screening tests, including, in the first two sessions a physical examination for the purpose of evaluating the effectiveness of the tests. Anyone found to have positive results was referred to his family physician or to an outpatient department for diagnosis and treatment. The physician was asked to report his final diagnosis. If no answer was received after a reasonable length of time, a nurse called to determine whether or not the patient had consulted him. If the patient had failed to see the doctor, a social-service worker followed up the patient.

Follow-up Study by Family Physician

A follow-up study of persons visiting the second session revealed that 62 per cent had been requested to visit their doctor. The family physicians reported on 1399 persons. Among 1252 persons, 2065 conditions were found, an average of 1.65 conditions per person. Of the remaining 147 persons checked by the family physician, 241 conditions could not be confirmed. The physicians also failed to mention 3098 conditions screened by the clinic on the same group of 1399 persons. It is not known whether these conditions were found to be present or so minor that they were not considered worth mentioning, or whether the physicians forgot to mention them.

In 1252 persons screened at the clinic and upon whom confirmatory reports were obtained from the family doctor, 848 conditions were found by the screening tests, 474 were found by the self-screener or history, and 365 were found by the evaluating physical examination in the clinic. Thus, a total of 1687 conditions were found at the clinic. If no clinic physician had been in attendance, 1322 conditions would have been picked up by the multiple screening tests and the self-screener combined. The family physician, on examining the patient and having available to him the results of the multiple screening tests and the self-screener, discovered 2065 conditions, or 743 more than were revealed by the multiple screening tests and the self-screener. Thus, approximately two thirds of the conditions that could be demonstrated by a combination of physical examination, laboratory tests and a self-screener can be found without a physical examination.

Since the primary objective of the clinic was to obtain medical care for persons who were apparently well but who had unrecognized conditions that required the attention of the family doctor, the fact that 64 per cent of the doctors to whom patients were sent reported their results is an indication that this objective was attained. Since there were, no doubt, some patients who saw their family physicians but whose visits were not reported to the clinic by either the doctors or themselves, well over two thirds of the patients who were asked to visit their family doctors probably did so. However, additional public education is required to bring an even higher proportion of patients into the doctor's office, for the ultimate measure of the success of the program must be the highest percentage of persons who see the doctor as soon as possible after referral.

The evaluation of the screening procedures utilized in the clinic indicates a high ratio of confirmation by the family physician of tests made in the clinic. When tests that experience has shown to give poor results are eliminated, 1 out of 2 findings were confirmed by the family physician; of the self-screener or history, 1 out of 7 conditions found were confirmed by the family physician under similar conditions. Since the family physician did not in the follow-up study evaluate all conditions reported to him by the clinic, it is possible that this ratio of confirmations could be even greater.

Table 1 presents an evaluation of the self-screener or clinical history, as a result of which we have come to the conclusion that a simplified history can be a valuable screening tool. However, the questionnaire is too long and may on occasion give falsely positive impressions. However, it is by far the cheapest method of determining the presence or absence of such conditions as overweight and underweight. Often the history can indicate certain chronic conditions requiring the physician's attention that cannot be picked up on any screening procedure thus far applicable on a mass basis.

Table 2 presents the evaluation of the multiple screening tests as confirmed by the family physician.

Table 1. *Evaluation of Self-Screener or Clinic History.*

Abnormality	Total Positive Screening Tests	Ratio of Confirmation by Family Physician*
Eye	355	1:12.7
Ear	320	1:11.9
Respiratory	178	1:19.7
Allergy	331	1:55.2
Hypertension	361	1:2.7
Heart	274	1:7.2
Hyperthyroidism	157	1:17.4
Arteriosclerosis	96	1:96.0
Varicose veins	397	1:8.6
Mouth	4	1:8.0
Hemorrhoids or rectal polyp	691	1:10.2
Other G. I.	213	1:10.1
Rheumatism	202	1:10.6
Skin	573	1:16.9
Diabetes	174	1:7.9
Breast	55	1:11.0
Cystocele	229	1:17.6
Female genitals	345	1:3.5
Prostate	75	1:6.3
Other male genitals	48	1:24.0
Hematuria	33	1:5.5
Other urinary	621	1:32.7
Overweight	538	1:1.8
Underweight	79	1:6.6
Organic nervous	204	1:68.0
Mental and psychosomatic	506	1:8.3
Liver	157	1:22.4

*Based on 1399 persons on whom family physicians reported.

On the average the ratio of confirmation for valid tests can be as high as 1:2.

Table 3 presents a comparison of the findings of the local family doctor and the clinic physician. The findings were made on the basis of the multiple screening tests, the history and the physical examination, and are merely given here for information and not for the purpose of evaluating the screening technics.

TABLE 2. *Evaluation of Multiple Screening Tests.*

FINDING	TOTAL POSITIVE SCREENING TESTS	SCREENING TESTS CONFIRMED BY FAMILY PHYSICIAN	RATIO OF CONFIRMATION BY FAMILY PHYSICIAN*
Anemia	108	70	1:1.5
Hearing defect	167	27	1:6.2
Heart disease	106	41	1:2.6
Hypertension	324	176	1:1.8
Albuminuria	72	11	1:6.5
Overweight	554	347	1:1.6
Tuberculosis of lung	3	1	1:3.0
Respiratory infection	32	10	1:3.2
Syphilis	7	5	1:1.4
Underweight	5	0	1:8.0
Visual defect	202	65	1:3.1
Diabetes	66	40	1:1.7
Glycosuria	87	6	1:14.5
Vaginal smear	15	2	1:7.5
Hematest	29	11	1:2.6
Hypotension	122	4	1:30.5
Abnormalities (shown by x-ray study)	121	17	1:7.1

*Based on 1399 persons on whom family physicians reported.

TABLE 3. *Comparison of the Findings of Family Physician and Clinic Doctor.**

DISEASE	FALSE-NEGATIVE SCREENING†	FALSE-POSITIVE SCREENING‡
	%	%
Anemia	59.5	1.2
Ear defect	10.5	0.7
Heart abnormality	14.2	2.9
Hypertension	17.8	3.6
Overweight	1.7	1.9
Underweight	23.5	0.0
Urinary abnormality	2.9	1.2
Cancer	57.1§	1.1
Tuberculosis of lung	50.0§	0.2
Other respiratory abnormality	17.5	0.8
Syphilis	0.0§	0.1
Eye defect	7.4	1.5
Diabetes	20.0	1.4
Glycosuria	25.0§	1.6
Breast condition	26.7	0.7
Cystocele	5.8	0.4
Other condition of female genitals	7.6	1.5
Thyroid abnormality	27.7	0.5
Varicose veins	1.5	0.4
Hemorrhoids	2.6	1.4
Other gastrointestinal disorder	2.7	4.1
Other disease of male genitals	7.1	0.5
Rheumatism	31.0	0.2
Skin disorder	6.0	1.1
Mental and psychosomatic disorder	27.0	1.8
Organic nervous disorder	30.0§	0.1
All others	57.6	1.9

*Based on the assumption that the family physician's diagnosis is correct. Unknowns are omitted.

†Negative screening test in a person with the disease.

‡Positive screening test in a person without disease.

§Rate poorly established; based on less than 25 cases.

COST

Table 4 presents an analysis of the cost of the health-protection clinic per session. The cost of this research program was much higher than that of a service program. The total cost per person in the first session, exclusive of central administration and research, was $10.04; in the second, it was $8.29. This included the expenses of the physical examination, the screening tests and the self-screener, as well as the salaries of all follow-up personnel and the amount for rent and supplies. The cost of applying the multiple screening tests, exclusive of the Papanicolaou test, ranged from $2.45 in the first session to $1.63 in the second. In a service program the use of the screening tests, of a modified history and overhead should be less than $2.00 per person. Table 5 presents the estimated cost of the application of each of the screening tests.

TABLE 4. *Cost of Health-Protection Clinics Per Person.*

ITEM	COST PER PERSON AT FIRST CLINIC*	COST PER PERSON AT SECOND CLINIC†
Physical examination	$3.97	$2.37
Rent and miscellaneous‡	1.52	2.11
Screening tests	2.82	1.83
Self-screener (history)	0.36	0.26
Receptionist and administrative nurse	0.39	0.29
Follow-up study and letters to physicians	0.98	1.43
Subtotal	$10.04	$8.29
Central administration	6.23	3.60
Total	$16.27	$11.89

*2620 persons.

†5057 persons.

‡In the second clinic rent included laundry, replacements and central supplies. The largest items of these were sheets, bathrobes, syringes, needles and rubber gloves.

TABLE 5. *Estimated Cost of Screening Tests.*

TEST	COST PER PERSON AT FIRST CLINIC*	COST PER PERSON AT SECOND CLINIC†
Vision	$0.22	$0.21
Hearing	0.17	0.12
X-ray study of chest	0.69	0.28
Urinalysis	0.26	0.17
Blood sugar	0.43	0.32
Blood Hinton	0.20	0.10
Hemoglobin	0.20	0.10
Papanicolaou	0.38‡	0.70‡
Hematest	0.02	0.03
Height, weight and blood pressure	0.26	0.20
Electrocardiogram	—	0.10
Totals	$2.83	$1.83
Cost per person (all tests without vaginal smear)	$2.45	$1.63
Weighted average cost (without vaginal smear)	$2.04	
Weighted average cost of self-screener	0.30	
Total multiple screening tests, including history	$2.34	

*2620 persons.

†5057 persons.

‡3.91, if cost is estimated per patient given test.

Table 6 presents the cost per condition found by the self-screener or history. These costs vary from a minimum of 59¢ for mental and psychosomatic conditions to a maximum of $2.46 for heart ailments, hyperthyroidism and diabetes. The cost of most conditions suspected of positive findings on the basis of history ranged between 72¢ and 89¢.

Table 7 presents the cost per condition screened for each of the multiple tests. Of particular interest were the costs of finding of a suspected pulmonary tuberculosis case ($96.62), a suspected case of syphilis ($51.62) and a suspected case of diabetes ($35.22).

Table 8 presents the cost per actual case confirmed by the family physician. It must be remembered that if reports were received from all physicians the cost per case would be much less than that illustrated by the confirmation of the findings of the clinic administrator, who performed this for the purpose of evaluating the effectiveness of the procedures. If all the cases reported to the family physician had been confirmed in the same proportion as by the clinic administrator, the cost per case confirmed by a final diagnosis would, of course, have been even less than that presented in Table 8.

TABLE 6. *Cost Per Condition Screened by the Self-Screener.**

CONDITION	WEIGHTED AVERAGE FIRST AND SECOND SESSIONS
Eye defect	$0.89
Ear defect	0.89
Respiratory disorder	0.89
Allergy	0.72
Hypertension	0.89
Heart disease	2.46
Hyperthyroidism	2.46
Arteriosclerosis	0.72
Varicose veins	0.65
Mouth disorder	0.72
Other gastrointestinal abnormality	0.89
Hemorrhoids or rectal polyp	0.65
Rheumatism	0.72
Skin disorder	0.72
Diabetes	2.46
Breast disease	0.72
Disease of female genitals	0.89
Other male genitals	0.72
Urinary disorder	0.72
Overweight	0.65
Underweight	0.65
Organic nervous disorder	0.72
Mental and psychosomatic disorders	0.59
Disease of prostate	0.89
Cystocele	0.72
Liver and gall-bladder disorders	0.89

*Includes all costs of administration, supplies, follow-up study and so forth, apportioned.

TABLE 7. *Cost Per Condition Screened by Multiple Test Alone.**

CONDITION	WEIGHTED AVERAGE OF FIRST AND SECOND SESSIONS
Anemia	$7.89
Hearing defect	4.50
Heart disorder	21.45
Hypertension	4.91
Renal disease	6.69
Overweight	2.76
Cancer of lung	25.82
Tuberculosis of lungs	92.62
Other respiratory disorder	4.67
Syphilis	51.62
Underweight	3.65
Visual defect	5.46
Diabetes	35.22
Glycosuria	4.77
Cancer of uterus	71.82
Other gastrointestinal disorder	7.13
Hypotension	3.60
Disease of bone and cartilage	3.22
Healed lesion (lung)	2.79

*Includes all costs of administration, supplies, follow-up study and so forth, apportioned.

SUMMARY AND CONCLUSIONS

It is the opinion of the Department of Public Health that in the near future all the research data that can be reasonably expected from such a study will have been obtained and that the final analysis of these data should be published so that it will be available, in accordance with the recommendations of the National Commission on Chronic Diseases, co-sponsored by the American Medical Association and other national groups.

As a result of the evaluation program, it is recommended that a physical examination not be considered a part of a screening program and that only procedures not giving a high proportion of false-positive or false-negative interpretation be used. It is further recommended that in a community screening program for chronic diseases recording of height, weight, pulse and blood pressure and temperature be omitted because experience reveals that these procedures are either unreliable in the clinic setup, disclose conditions that are already known, or can readily be determined at the time the person sees his doctor. Examination of the urine may be omitted, unless doctors in the area wish it to be made, since this is a procedure that can be carried out in the doctor's office. Hearing tests can be omitted since in most examinations the confirmation of a screening test is lacking, unless special arrangements are made for the follow-up study of positive screening results.

The vaginal smear could also be omitted — first, because of the high cost, secondly, because of the small number of cases yielding a positive result and, thirdly, because of the cumbersome arrangements for such a procedure in a clinic of this type.

The following procedures may well be utilized in a program integrated with the mass tuberculosis

TABLE 8. *Cost of Screening Individual Condition.**

CONDITION	DIAGNOSIS CONFIRMED BY FAMILY PHYSICIAN		CONDITIONS REPORTED BY CLINIC ADMINISTRATOR	
	1ST CLINIC SESSION	2D CLINIC SESSION	1ST CLINIC SESSION	2D CLINIC SESSION
Anemia	$32.81	$18.55	$14.29	$7.33
Heart disease	26.97	46.94	9.65	11.23
Hypertension	11.05	18.37	4.73	5.76
Hypotension	8.37	17.29	21.43	34.41
Visual defect	23.93	26.60	3.95	6.01
Hearing defect	30.69	34.15	4.55	5.07
Overweight	10.20	13.35	3.78	4.23
Underweight	7.84	14.02	3.55	3.87
Tuberculosis of lung	467.78	727.35	41.13	85.88
Syphilis	63.49	116.20	45.79	50.36
Diabetes	52.36	67.44	17.06	17.25
Glycosuria	117.93	81.12	90.83	29.56
Healed lesion of lung	8.57	11.12	3.57	10.48
Disease of bone and cartilage	23.03	25.46	8.66	8.09

*Includes all costs of administration, supplies, follow-up and so forth, apportioned.

survey now usually carried out by state and many city health departments:

A 70-mm. photofluorographic plate of the chest for the detection of pulmonary tuberculosis, pulmonary tumors and other defects of the chest.

A blood test for the detection of diabetes, anemia and syphilis.

Screening for the detection of visual defects.

An electrocardiogram consisting of one lead to pick up approximately 50 per cent of the cardiac lesions that are susceptible of being found in a 12-lead tracing.

A modified self-screener or history for the detection of some other chronic conditions and for verification of the presence of some of the chronic diseases that can be picked up by the screening tests.

A hematest for the detection of blood in the feces may be included if desired.

Thus for an expenditure of less than $2.00 in such a mass screening survey it will be possible to apply these procedures instead of merely screening by a single test of a photofluorographic plate at an average cost of 75¢ per person, or in a syphilis case-finding program the cost of over $1.00 per person screened. In other words, for about double the cost of conducting a tuberculosis case-finding program in which other conditions of the chest are picked up on the photofluorographic plate, it is possible to add tests that will screen the same person for additional defects.

In conclusion we should like to express the gratitude of the Department to the many physicians who have so ably co-operated in this program and to quote from President Arthur W. Allen's Address to the Council of the Massachusetts Medical Society on May 23, 1949:

> You have approved of the recommendation of the Committee on Public Health to appoint a subcommittee for the purpose of developing pilot diagnostic clinics. . . . The beneficial effects already demonstrated by the Tuberculosis League, the State Cancer Society, the Committee on Diabetes and the Heart Association are well known to you. If the mechanism of clinics for general examinations can be satisfactorily worked out for all concerned, we shall have made a great forward step. It is requested that those of you who are skeptical of such a venture offer constructive criticism and lend your aid to this endeavor.

The Department is of the opinion that Dr. Allen's prophecy about the taking of "a great forward step" has been accomplished: not only have we induced apparently well persons who had unrecognized defects to seek early diagnosis and treatment from the family physician, but also we have been able to evaluate many of the mass screening technics — some obviously have to be discarded, but others have proved their value. From time to time other tests may be developed and should be critically evaluated. The medical profession is to be congratulated on participating in this evaluation of the mass screening program, and in the health education of the public designed to persuade people to seek early medical care from their family physicians.

IMPORTANT FEATURES IN AN INDUSTRIAL MEDICAL PROGRAM*

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A MEDICAL program applied to a specific industry should be tailored to fit the needs of that industry. The nature of the industry, its size and its geography are important factors in the selection of the parts of a health program that deserve special emphasis. The operations of a chemical plant, for example, require careful attention to the problems of toxic exposure, whereas a heavy manufacturing industry has as its major problem, the prevention and care of trauma. However, people are still hurt in chemical and poisoned in heavy manufacturing industries, and these hazards exist regardless of whether the industry employs 100 or 20,000 people. Any medical program, therefore, should devote attention to the prevention, detection and adequate therapy of both occupational illness and trauma and should not concentrate on one to the exclusion of the other.

There is wide variation in the type of medical services provided within industry today. Many smaller plants of 500 employees or less have no medical or nursing services whatever. This in itself is an important challenge since approximately 70 per cent of all industrial workers are employed in these small plants. Time does not permit me to discuss the various aspects of the over-all problem, except to say that there are several satisfactory approaches available. In the industries that provide medical services for their employees, there is a wide variation in the scope and adequacy of such services. At one end of the scale are the plants in which first-aid services only are provided. At the other end are the industries — and there are a few — that provide complete medical care not only to employees but also to their families. I think it will be agreed that the former is entirely inadequate and that the latter exceeds the principles upon which an industrial medical program is based; also, with the emphasis so heavy on treatment, the important preventive and educational aspects of health are apt to suffer.

An industrial medical program, however, must embrace considerably more than the problems of

*Presented at the annual meeting of the Massachusetts Medical Society, Boston, May 21, 1952.

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occupational disease and injury if it is to be of maximum service to the worker and to the industry as a whole. The World Health Organization has defined health as a state of complete physical, mental and social well-being — not merely the absence of disease. Johnstone,* in his book *Occupational Medicine and Industrial Hygiene*, states in part as follows:

> We must cease looking upon the working man as an 8 hour a day problem in which only his occupational environment is of concern. No longer can the industrial laborer be classified as a mechanical component of the machinery of industry. We have realized that the worker is a member of society, whose working, playing and living is a 24 hour problem. Such a concept entails consideration of all his faculties — his capacities as well as his deficiencies. It includes a program which will safeguard his hearing, his vision, his nutrition, his skills, aptitudes, experience, intelligence and emotions. . . . Man's work is related to his home, his social environment, his philosophy, his economics, and his happiness. . . .

That these statements are not merely speculative is demonstrated in part by Figure 1. As shown, the

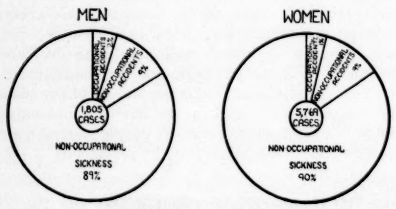

FIGURE 1. *Distribution of Lost Time from Disability due to Occupational Accidents (One Day or More) and Sickness and Nonoccupational Accidents (Eight Days or More) in 1951.*

greatest percentage of absence from disability is due to nonoccupational illness — conditions that are the result of influences outside the working environment. An industrial medical program should devote much of its attention and activity to this phase of health, although the still existing but certainly less significant problems of occupational illnesses and injuries should, of course, not be overlooked.

Medical Services in Industry

For many years the American College of Surgeons successfully carried on a program of evaluation of medical services in industry. Realizing that in recent years the subject of industrial health and health services has become large and complex, the College did not consider itself equipped to continue with this survey service. As a result, approximately a year ago this program of evaluation of medical services in industry was transferred to the Industrial Medical Association to be carried on under the auspices of its American Foundation of Occupational Health. The first thing the Foundation undertook was a program for re-evaluation of industrial medical practices today in an attempt to arrive at some sort of up-to-date standards. To this end the medical establishments of numerous industries have been physically inspected and surveyed. In addition self-surveys were more recently mailed to the medical directors of 100 industries whose medical departments were familiar to one or more of the Foundation Trustees and who are known to have better than average medical programs. The plants surveyed were selected so as to provide a sampling of industry by size, by type and by geographic distribution. In the analysis of these returns, particular attention is given to the following subjects: medical policy; person to whom medical director reports; qualifications of physicians; duty hours of physicians; scope of service offered (first aid, preplacement physical examinations, periodic physical inventories and supervision of health, sanitation and industrial-hygiene problems); facilities for complete diagnostic examinations (personnel, space and equipment); medical records; management's attitude toward medical phases of human relations; attitude of management toward medical department; and attitude of employees toward medical department.

The coverage of industries is indicated in Table 1.

Since the original 100, 50 more industries have been added, but these returns are not as yet in. The returns from the 100 industries listed in Table 1 were also received too recently to permit detailed analysis of all items mentioned above, but preliminary study has served to bring out and to confirm impressions of some important features, which are described below.

TABLE 1. *Nature of 100 Industries Covered.*

Industry	Number	Industry	Number
Chemical	11	Electric lamps	2
Petroleum	6	Converters	1
Steel	8	Food	2
Paper	2	Explosives	3
Drugs	6	Metal	2
Automotive	4	Utility	3
Die casting	9	Meat packing	3
Electronics	2	Insurance	1
Research	2	Rubber	1
Holding company	1	Textile	3
Printing	4	Refrigerators	1
Heavy machinery	14	Aircraft	3
Engines	1	Shoe manufacturing	1
Plastics	1	Electrical supplies	3

Medical Policies

Of the 100 industries surveyed, 75 per cent have written medical policy. Some other industries — not included in the self-survey — have a policy that is incorporated as a part of industrial-relations manuals. In most industries these portions relate only to a statement of the procedures required by

*Johnstone, R. T. *Occupational Medicine and Industrial Hygiene.* 604 pp. St. Louis: The C. V. Mosby Company, 1948.

compensation and other laws and do not contain the over-all policy. It is my belief that a written medical policy is valuable. It serves to acquaint management, employees and the medical and nursing staffs with the specific objectives, procedures and practices of the medical department. A written medical policy can also be of help in straightening out points of contention that occasionally arise between a private practitioner and the industrial medical department or between groups within the industry itself.

Reporting Level of Medical Director

In 77 per cent of the 100 industries surveyed, the medical director reports either to an operating or to a personnel executive. When the medical director serves in a part-time capacity, the reporting level varies. It is my opinion that in any industry in which the medical director devotes full time, he should report to an officer of the company, whether in operations or in personnel. This is a more important point than might appear at first glance since the medical department is in a unique position. It is, or should be, a service organization existing for the benefit of all employees, and to be effective it must be neutral and impartial. Moreover, the medical department has certain responsibilities toward both management and employee. However, when differences of opinion arise, if the medical department has a reputation for fairness and impartiality and if its opinion is based on sound medical reasoning, such differences can usually be easily resolved to the satisfaction of all concerned. The medical director, nevertheless, must report sufficiently high in the organization to preserve his position of neutrality. If he reports to someone in the industrial-relations department, he is likely to be identified, even though mistakenly, as a tool of management. He is then apt to be distrusted by labor and may consequently be hindered or even blocked from undertaking many constructive health activities. Direct reporting to an officer of the company is also important in that it enables the medical director to submit recommendations involving practices or possible modifications of medical policy at a level where they may receive the proper executive consideration and to prevent them from becoming lost in the morass of organizational structure.

Medical Records

In 99 per cent of the companies surveyed, medical records are considered as confidential information and are preserved in the medical department. Exceptions, which are made by law, usually concern findings on preplacement medical examination and occupational diseases or injuries. Aside from this, the principles of physician-patient relation apply as in private practice. All clinical records should be maintained in the medical department and should not leave it except under subpoena. If management requests information on an employee that it is properly entitled to receive, the medical director or plant physician can give this information from the record, which should, however, remain in the department. Except for the employee's attending physician or attorney, requests for medical information from outside persons or agencies should be honored only with the written authorization of the employee concerned.

Medical records should be adequate and complete. To most physicians this sounds as though it should be self-evident. However, it is surprising how often when an attorney has occasion to address a group of physicians about the medicolegal phases of their work, he mentions the inadequacy of medical records. Good medical records are essential in the interests not only of the patient but also of the industry when occupational disease and injury are involved. They are also important to the physician's interests.

A physician does not acquire immunity against personal lawsuits for malpractice simply because he performs services within an industrial medical establishment. He can be sued personally. It is well, therefore, for him not only to maintain good records but also to carry personal malpractice insurance. Many physicians do not realize that malpractice insurance carried by the industry protects only the industry against suits for acts committed by its agents. It does not protect the physician who is alleged to have committed the act.

Professional Features

Thus far I have discussed a few of the important administrative features of medical-department programs. However, certain professional activities have also demonstrated their value in industrial medical programs in which these procedures have been properly utilized. Any health activity must render a real service to the employees both individually and as a group if it is to be effective but the benefits derived by the industry itself may be substantial. (Although the masculine gender is used I wish to emphasize the fact that the subject is applicable to both sexes.)

Preplacement Examination

Originally objected to by many labor organizations on the assumption that its only purpose was to deny employment to workers with physical abnormalities, the preplacement examination is today quite generally accepted. This examination has several purposes. It attempts to effect placement of an applicant on a job within his physical limitations so that he will not constitute a hazard either to himself or to others. This examination also helps to prevent the introduction of communicable disease into the existing employee group.

It provides the starting point for future health supervision during the term of company service. Frequently, this examination is the first the worker has had for many years. As a result, many minor and incipient major abnormalities are detected and brought to the applicant's attention at a stage when they may be more easily corrected. I believe that a urinalysis should be routine. If convenient, a chest x-ray examination should be made a part of the preplacement procedure. In the dusty trades, of course, it should be routine.

Periodic Health Examinations

The rationale of this activity is based on two considerations: the earlier a disease is detected, the more amenable it is to cure or control; and most illnesses, other than acute infections, are asymptomatic in the early stages and the average patient does not as a rule consult his physician unless he is experiencing symptoms of some type. The periodic examination of a person presumably well, therefore, has real merit in the early detection of disease. It should be mentioned that the periodic health examination, if properly performed, is more exhaustive than the various laboratory screening procedures. To be effective, the examination should be voluntary on the part of the patient lest important symptoms be deliberately concealed. The findings, of course, should be confidential and should be reported only to the patient or to his personal physician.

Diagnostic Examinations

In essence, these cover much the same ground as the periodic examination. However, the patient usually presents himself because of symptoms. It is recognized that, except in large industries, medical-department facilities for complete diagnostic work-up may be limited. However, it is well to remember that the history and physical examination are still the most important parts of any medical work-up, and a good physician with a flashlight, tongue depressor, stethoscope, blood-pressure cuff and rubber glove can cover most of the ground fairly well. The tendency may be to overaccentuate the importance of laboratory procedures. In medical establishments not equipped for various laboratory procedures, such determinations can usually be obtained, when indicated, through the medium of an outside commercial laboratory or by referral to a hospital laboratory.

Disability Evaluations

This type of examination is usually carried out on patients who have convalesced from either an injury or a prolonged illness. A check on such a patient's physical and mental status before his return to work serves to determine whether or not further convalescence is advisable. It also reveals any residual disabilities or impairments. It serves to prevent the worker from returning to a job assignment that is too exacting for his physical capacities at the time of his return. When a permanent disability is involved, the medical department is given an opportunity to arrange with the operating department for job reassignment.

Treatment

An industrial medical department should not engage in definitive treatment of nonoccupational conditions — this, in effect, would be entering into competition with the private practitioner in the treatment of his patients. In such conditions, the industrial physician should co-operate in every possible way with the employee's personal physician and endeavor to assist him, on request, in his efforts at diagnosis, treatment or rehabilitation. Treatment of nonoccupational conditions within the industrial medical department, therefore, should consist only of first aid and initial or emergency care. Certain preventive procedures that are generally recognized as good public-health measures may, of course, be carried out. Often, it is convenient for employees to receive injections at the plant medical department in the treatment of various ailments — examples are liver extract in the therapy of certain anemias and pollen extracts for hay fever. This type of injection therapy should be done only upon the written request and the written specifications of the attending physician. In such cases it is also well to remember to refer the employee to the attending physician periodically for his recommendations regarding the continuation of such treatments.

Use of Consultants

No matter how elaborate and well staffed an industrial medical department may be, it cannot hope to have experts in all the specialties on the premises. Nor can the industrial physician, or any other physician for that matter, become an expert in all the medical specialties. Solution of this problem lies in the utilization of outside consultants when special problems arise. It is usually the better part of wisdom for the physician to recognize his limitations and not to hesitate to avail himself liberally of the services of well recognized consultants.

Advice on Personal Problems

An employee, if he has confidence in his medical department and if he has not been subjected to cursory attention in previous contacts, usually has no hesitancy in seeking advice on health problems of his own. The same employee, however, may be worried over a family situation. This may be senile deterioration in an aged parent, addictive drinking in his spouse, possible delinquency of one of the children or some other delicate family problem. He may be embarrassed or ashamed to seek outside advice, or he may be eager to receive such advice without knowing where to turn. He may brood

about a problem long enough for a disabling anxiety state to develop. Also, such a situation is frequently responsible for serious accidents, especially when such a person is working around moving machinery. The medical department should encourage visits from employees with problems of this sort. If confidences are maintained and if understanding listening is employed, much helpful advice can usually be given, with resultant benefit to the employee concerned as well as to his family. It is worth while to note that men are generally more willing to pour out personal troubles to a woman, and in this connection the industrial nurse can be of enormous help.

Health Education

Figure 1 demonstrates that approximately 90 per cent of disability absence is the result of conditions not directly related to the working environment. Since the worker's free time is under no environmental control other than his own, approaches to this problem must be by methods calculated to influence personal behavior. Methods that are applicable on a mass basis should be utilized. This type of activity, which is usually referred to as health education, may be undertaken by means of group lectures, posters on bulletin boards, pamphlets and sound films on health matters, company-sponsored courses for employees on nutrition, first aid, and other health and safety measures. General articles on health and medical subjects in company magazines are also important mediums for health education, and if used with discretion, they can be most effective.

Rehabilitation

This phase of medicine remained quite generally in the background until interest was stimulated as the result of the military program of rehabilitation of casualties of World War II. Although rehabilitation today is a familiar word, many still tend to think of it in terms of a large establishment equipped with pulleys, weights, bicycles, treadmills and other paraphernalia, and replete with physiotherapists, occupational therapists, psychologists and vocational-guidance counselors. There is no question about the valuable functions that such establishments perform, but rehabilitation should not be reserved exclusively for patients with major amputations, the partially paralyzed and other seriously crippled persons. In industry, especially, rehabilitative procedures of lesser degree are of considerable value. In cases of relatively long illness, an understanding attitude together with reassurance and encouragement on the part of the physician or nurse can accomplish much. On the physical side, rehabilitation is often indicated in injuries ordinarily considered minor. For example, quadriceps-muscle-setting exercises should be instituted at once when any disabling injury involves the knee joint. Unless exercised, the quadriceps-muscle group atrophies rapidly in such a situation, but even today many patients exhibit a limited capacity for work for extended periods because of a weak quadriceps muscle long after the knee joint itself has returned to normal. Although it may be difficult in smaller industries, it is usually possible in larger industries to arrange for an injured employee not yet ready for his regular work assignment to return to some type of modified assignment and even to an assignment where his work will require active exercise of the injured part if such exercise is indicated. However, the foreman or supervisor should not assume full responsibility for such an arrangement, because he does not know the employee's work capacity. The employee usually does not investigate such a possibility, for he does not know what assignments may be available. It is therefore up to the industrial physician to initiate the arrangement. In closing, it should be emphasized that the features mentioned should be possible of adoption to some degree at least in any industrial medical department where the services of a physician are available, regardless of the size of the industry.

That not only management and the industrial physicians but also the labor organizations are interested in promoting industrial-health programs is evident in many quarters today. A recent issue of the *Massachusetts Health Journal* is devoted entirely to health services in industry. In that issue — the following paragraphs appear as part of an article by George F. Markham, New England educational director, International Fur and Leather Workers Union:

> Regardless, however, of what the employer's motivation may be for urging an industrial health program, unions should encourage it and see that the workers' rights are protected.
>
> The employer is concerned because 2,000,000 man-years of work are lost each year by illness of industrial workers. The union should be interested because a real health program can reduce the workers' suffering from disease, save them money, retain their earning capacity for more years, and benefit their families and communities.

MEDICINE + INDUSTRY = OCCUPATIONAL HEALTH*

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THIS is a dynamic world. Within the span of a single lifetime the machine age has transformed this country from an essentially rural people to one of urban habitat. With this fundamental change, certain significant social and economic trends have resulted. Industrial medicine, as a special field of medical practice, affords organized medicine a direct opportunity to experience and study the conditions that are influencing these current unprecedented trends. That these changes may move in the right direction, it is imperative that all branches of medicine participate in their interpretation and guidance.

The social and economic issues raised during the past few years have sharply focused the attention of medical and nonmedical people on the tremendous strides that have been made in medical care since the turn of the century. All admit that, in spite of these significant gains, there is much left undone. Attention need only to be called to the problem of the "common cold" to suggest the challenges that lie ahead.

Progress naturally heralds shifts in emphasis. History bears stark evidence that medicine has proved no exception to this basic principle. So, today, physicians are grappling with the concept of health just as a few years ago they were struggling to gain an understanding of disease. The practice of industrial medicine with the establishment of industrial health services affords an exceptional opportunity for the medical profession to become oriented to health as a positive concept through participation in the practice of preventive medicine. Industrial medicine is not a restricted specialty and, if properly practiced, approaches a return to the concept of the family physician, who is concerned with the patient as a whole and all that affects his welfare.

The growth of industrial medicine was given marked impetus during the second decade of this century by the passage of the workmen's compensation laws and the rapidly expanded industrialization necessitated by World War I. This entire movement has been almost entirely limited to the larger industries, and until recently the reparative and curative phases of medical practice were stressed, the preventive and constructive aspects taking a minor role. World War II set the stage for industrial physicians and management to recognize that a change of outlook was necessary if they were to meet production schedules. Whereas emphasis had formerly been placed on "what was wrong," this new philosophy stressed "what was right." This shift in emphasis has been an important factor in the expansion of existing industrial health services and in the installing of new ones.

With expanding industrialization and growing labor shortages, it must be recognized that, although the larger industries have, for the most part, developed adequate health services for their workers, almost nothing has been accomplished for the small plants. The magnitude of this problem is suggested by the fact that 99 per cent of this nation's employers and 60 per cent of the industrial work force are located in establishments of under 500 employees. Few of these plants or their employees are experiencing the constructive benefits of in-plant health services.

Further to delineate the scope of this problem and to indicate its significance to the medical profession, the American Academy of General Practice reported in 1950 that a survey of "Distribution and Type of Practice of Members" revealed that 95 per cent of their membership cared for industrial cases. This emphasizes the key role that the general practitioners have in accepting the responsibility for adequate industrial health maintenance.

During the past few years there has been an urgent need on the part of management, workers and the health professions for the development of a basic plan that will afford the employers of small groups an opportunity to bring the advantages of an adequate health service to their employees. To date, progress has been slow and often discouraging mainly because of the lack of experience on the part of both management and physicians. There is need for the development of an intensive and continuing educational program for small-plant industrial health services that will encourage management to support such services, and to assist, effectively and ethically, the general practitioners who participate in industrial activity in the organization and direction of these services. Health maintenance, which is the keystone to the arch of successful industrial medical practice, effectively complements the already existing traumatic and curative services rendered to most employed groups.

Employers and employees in small plants have every opportunity to match the high caliber of services that exists in the full-time occupational health departments of the larger industries. Obviously, the extent of services rendered may vary widely, depending upon many variables, including the number of employees, geographic location and industrial hazards.

*Presented at the annual meeting of the Massachusetts Medical Society, Boston, May 21, 1952.

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The extension of effective health services to small plants is bound to result in benefits that both employer and employee can share. The employee will have more opportunity to discuss health problems, both his own and those of his family, with someone who is interested and who is competent to advise him. He is not expected to be disabled and in need of treatment before his problems are given consideration. Because his illnesses are recognized early, treatment becomes more effective, and disability is avoided altogether or at least shortened. He is encouraged to seek advice promptly because it is more readily available and because he understands that its purpose is to keep him well and on the job.

The employer, since he provides the financial support for the health services, has a vital interest in how these services will benefit him. He knows that the success of his business is gauged by his ability to produce at a profit in today's highly competitive market. Healthy people are productive people, and in small industry this correlation is most significant because of the intimate interdependence that exists between management and employees. The proper selective placement and health maintenance of his employees, to the same degree and with the same care that he has learned is necessary with his machines, will keep his present workers on the job and attract desirable new employees. Every employer is conscious of his community obligations; he must not fail to recognize the contribution of an adequate industrial health service to improvement of health in the community.

That the community benefits from increased efforts at keeping workers healthy need hardly be emphasized. The large number of communities dependent on small plants for their economic existence makes it evident that the real opportunities for raising the health standards of the entire nation lie in these efforts to promote the health of workers in small plants. The physicians of the community can no longer be concerned solely with the sick and the injured but must accept their responsibility for the maintenance of a healthy industrial population.

The cornerstone in building a preventive medical program in industry is a physician with real interest in applying the principles and procedures of prevention to the medical problems peculiar to industry. His personality should be such that people will bring their problems to him as a physician interested in the human side of medicine, as contrasted to the mechanics of medical care. He should have full knowledge that his function embodies the development of an effective employee health program that goes well beyond the treatment of injuries and the usual medical complaints. Thus, his approach to in-plant medicine is preventive medicine, which has two major goals, primary prevention and secondary prevention. Primary prevention means keeping an illness from occurring, and secondary prevention means early detection followed by elimination or modification of personal and environmental factors, which if left uncontrolled would probably lead to more serious illness or injury.

He should interest himself in all problems of the plant relating to employee health. These vary in different industries, involving toxicology, trauma, emotional and morale problems, aging and disabilities due to chronic illness. If he has had no previous experience or training in industry, he should be interested in developing his knowledge in this field by learning the many aspects of industrial medicine that are not usually regarded as being of a medical nature, such as the physical and emotional demands of jobs, labor relations and industrial hygiene and safety. He must recognize that the primary concern of medicine in industry is the relation between occupations and health, which necessitates an understanding of the job, the man and the work environment. The work environment is a segment of the total community environment, and the interrelations existing between them affords the plant physician an excellent opportunity to utilize the available services to the mutual benefit of industry and the community. Since many aspects of industrial medicine are essentially nonmedical in the usual sense of the word, industry should play an active role in orienting physicians in how industry is organized and functions, and thus bring to the physician a knowledge of industrial relations, insurance experience, compensation practices and the like.

Although the physician should be essentially the internist or general practitioner type, he should have competence in the handling of minor trauma. Unless he has special surgical training, major cases should be referred to specialists in the field, and their caliber, interest and orientation to the problems of industry are important. He should have a knowledge of available community facilities for proper referral of employees and should have or build a rapport with the community physicians.

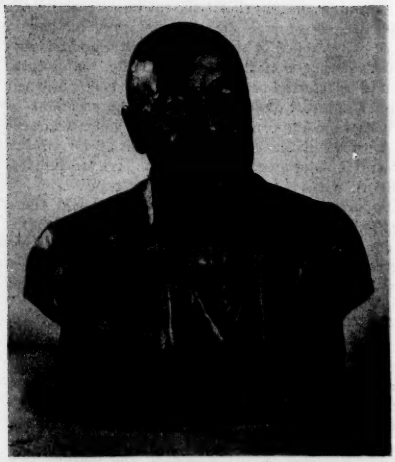
To further his knowledge in this special field, there are several professional organizations and publications with which he should become familiar. He should participate in local meetings and, when possible, in the national conferences on industrial medicine.

Time is the most important tool a doctor has, and he cannot be effective unless he is brought into the plant for enough time to meet its medical needs. His essential role in industry is planning a program and furnishing service. He can do neither adequately without sufficient in-plant time.

The industrial physician's relations with his fellow practitioners, management, the workers and the community are important. There should be free and unrestricted intercommunication between the industrial physician, the individual worker

concerned and the private practitioner of the worker's choice. The industrial physician should be responsible to management at the policy-forming level. He should discuss his findings with the worker involved, and when problems of health or medical care are discovered appropriate referrals should be promptly made to the family physician. All medical information must be held in strict confidence, the employer being furnished only a medical opinion or rating of employability and recommendations for suitability of placement. The confidential relation between physician and employee should be maintained at all times. Access to any worker's health record should be given only on the written request or consent of the worker or when required by law. An industrial physician should not take advantage of his industrial connection to solicit for the benefit of his own private practice.

In conclusion, it is well to recognize the fact that labor, management and government are becoming increasingly conscious of the values inherent in good health. They are striving for a successful formula or system that will give them what they think they need. This is medicine's golden opportunity to assume leadership and guide the people of this country toward a solution of this problem in the typical American, free-enterprise way. Industry affords the perfect setting; medicine is potentially capable of meeting this challenge, and solution lies in the development of adequate occupational health programs in plants, large and small, throughout the width and breadth of this vast industrial empire.



MASSACHUSETTS PHYSICIANS ART ASSOCIATION

8 Dr. Frederick J. Cotton

Sculpture by

SIDNEY C. WIGGIN, M.D.

President, Massachusetts Physicians Art Association

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MEDICAL PROGRESS

OCCUPATIONAL MEDICINE

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THIS report deals with the effects of chemical, bacteriologic and physical agents of known or possible hazard to the worker. The title "occupational medicine" was chosen as a description broad enough to include the laboratory worker, the farmer and the housewife. This is in contrast to the title "industrial medicine," which usually refers to groups of individuals engaged in operations of manufacturing such as ore smelting, storage-battery manufacture and sandblasting in a foundry. By this definition, consideration of the vast field of occupational health, which includes rehabilitation, preplacement examination, occupational psychiatry, the problems of the cardiac patient and the older worker in industry and proper medical service in large and small plants, will be left to another report. Dermatoses of occupational origin are also omitted from this discussion. The recent medical literature dealing with etiology, pathogenesis and treatment of occupational disease has been reviewed. Much of this information is not regularly available to the physician because it is published in the specialty journals, such as the *Archives of Industrial Hygiene and Occupational Medicine*, the *British Journal of Industrial Medicine* and *Industrial Medicine and Surgery*, to which the interested reader is referred.

OCCUPATIONAL INFECTIONS

Anthrax continues to be a real industrial problem, and because of failure to make a prompt diagnosis the mortality remains too high. Wolff and Heimann,[1] of the United States Public Health Service, made a survey published in 1951. They found highly infective carpet wool to be the current source of a recent increase in human anthrax. Goat hair and skin were shown to be a cause of the disease in the leather industry. Kolb and his associates[2] presented a full and useful discussion of methods of disinfection of anthrax-infected animal products. It is interesting to know that the British Government disinfects yearly 7,000,000 pounds of animal material, which includes all imported wool, hair and bristles, at its station in Liverpool. Because of the many ports of entry and varying state regulations, it appears that central disinfection for the United States is not practical. Kolb et al.[2] propose the use of methyl bromide as an agent that is sporocidal and not harmful to the animal product intended for industrial use. Since methyl bromide is highly toxic to human beings, however, the application of this plan in industry will require attention to protection of the workers involved. Proper protective clothing and cleanliness as provided by the Massachusetts Division of Occupational Hygiene[3] apparently do not provide enough protection, for cases continue to appear. There should not, however, continue to be mortality or serious morbidity if, as the Massachusetts regulations go on to point out, all suspicious lesions must be seen by a physician engaged for this duty who has "a thorough knowledge of the problems of anthrax." Clarke[4] reports successful treatment of anthrax with chloromycetin (Chloramphenicol) in 4 cases. Gold and Boger[5] describe good results with aureomycin, chloromycetin and terramycin, of the newer antibiotics. Penicillin has been shown to be capable of destroying anthrax when used in sufficient dosage.

Abrams and Warr,[6] discussing occupational diseases of animal origin, demonstrate that there is a higher attack rate of brucellosis among urban meat handlers than among farmers. They cite the figure of 60 per cent of 268 proved cases in Minnesota as being of occupational origin. A recent case of brucellosis in a twenty-year-old meat handler seen at the Massachusetts General Hospital, who for nearly two years had been regarded as a psychoneurotic patient, emphasizes that this fact is not generally known.

Q fever has been described by Bell et al.[7] among workers in dairies, livestock yards and meat-packing and fat-rendering plants in Southern California. Sigel and his associates,[8] by the complement-fixation test, identified illness among men working at scouring, blending and dying wool and in a goat-hair-processing plant as suffering from Q fever. The organism, a rickettsia (*Coxiella burnetii*), caused a mild grippe-like infection called "flu" by the men. Out of 152 tested, 48 had titers above 1:32, and 29 had titers above 1:128.

Skinner and Maloof,[9] discussing the hazards of men and women engaged in the fish industry, describe the long-known and troublesome erysipeloid. The organism *Erysipelothrix rhusiopathiae* is present in the "gurry," the fish remains that are used for animal feed. A violent lymphangitis and frequently a disabling arthritis follow a slight wound contaminated with the organism. Such infection yields to prompt use of the antibiotics.

Skinner[10] has reported a fatal case of coccidioidomycosis in a worker in a Massachusetts wool-

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scouring plant. The worker had been sorting wool from California.

Sporotrichosis in a sewage digger was observed by Tulipan and Muskatblit.[11] The patient responded promptly to potassium iodide and aureomycin. The authors report that agricultural workers, berry pickers, grain-mill workers and florists may contract trichosis.

Sulkin and Pike[12] cite an extraordinary number of infections in laboratory workers, reaching 1342 documented in 1950. Tularemia is mentioned as "approaching 100 per cent" in incidence.[13] The recent literature yields records of 3 cases of laboratory workers infected with the virus of Newcastle disease,[14] 6 cases of Coxsackie virus infection[15] and 1 laboratory infection with *Leishmania donovani*.[16] More scrupulous attention to protection for trained workers seems urgently needed.

Occupational Cancer

Considerable public attention has been aroused in the effort to increase understanding of the malignant process. An increasing amount of work is being done to detect the role of industrial operations as a single or partial etiology. It is tempting to believe that the inhalation of certain chemical fumes from an industrial operation by a worker or from the stack of an adjacent plant by a neighbor may be responsible for the documented increase in pulmonary carcinoma. Hueper,[17] participating in the United States Technical Conference on Air Pollution, presents a long list indeed of carcinogenic agents of possible importance as neighborhood contaminants. He properly qualifies this list by pointing out that significant quantitative levels must be demonstrated before etiologic correlation is made. Hueper[18] also discusses the currently accepted hazards of cancer in American industry. Not all workers in this field will agree with him that every relation he has suggested has been finally and satisfactorily demonstrated. However, in view of the seriousness of the problem, it is doubtless wise to stimulate accurate study of all suspected occupational carcinogens.

Scrupulous attention to the collection and interpretation of epidemiologic data is the present need. Mayers[19] has recently described such a study of occupational factors undertaken in New York State, using 6000 male adults under seventy-five years of age admitted to the Roswell Park Memorial Institute in Buffalo for diagnosis and treatment of suspected malignant lesions. She discusses the difficulty of getting reliable information. Various states are doing similar studies through their health departments. Merrill[20] describes the New Jersey survey, in which the percentage of total cases is charged to certain selected occupations and affected organ sites are shown by occupation. Mayers emphasizes the difficulties in interpretation of such data because of the many variables involved. She also points out the expense and time such work demands and wisely suggests that with this backlog of experience collection of data started now may give reliable answers fifteen or twenty years hence.

Heller,[21] in reviewing the chemical carcinogens presents data, recent and historic, to demonstrate the origin of cancer of skin, nasal sinuses, lung, bone and blood-forming organs from a variety of occupations. He discusses the use of experimental animal work in alerting industry to potential carcinogens. For instance, chloroform, selenium, beryllium, carbon tetrachloride, various aromatic azo compounds and carbamates used in the manufacture of rubber have produced tumors in small animals. Heller believes this should, as he puts it, "raise our index of suspicion in relation to the human problem." He points out the lack of relation between toxicity and carcinogenicity; therefore, control of the toxic level of benzene, nickel carbonyl or radiation may not prevent its production of malignant tumors at a chronic, low level.

The petroleum industry has done careful work in the study and prevention of skin cancer among its workers exposed to high-boiling, catalytically cracked oils. This is reported in a symposium[22] worth reviewing as an illustration of what preventive teamwork can do with current knowledge. It has long been known that high-boiling petroleum products used for lubricating oils are carcinogens, but the operations involved in the manufacture of catalytically cracked oils is relatively new to industry. This example demonstrates the need for continuous vigilance to prevent occupational cancer.

Knowledge of cutaneous cancer from certain soots dates from the beginning of the nineteenth century, when Percival Potts described the scrotal cancer of English boys used as chimney sweeps. Ingalls,[23] in 1950, made a careful study of a carbon-black manufacturing plant and found that among 476 workers on an annual average only 0.21 thousand per work year showed malignant change of the skin. This is low by comparison, and, as Ingalls says, the carbon-black worker with required daily showers and modern engineering protection does not face the same hazard as the English chimney sweeps did. Heller suggests that Ingalls' study is short term and that a final answer requires fifteen to thirty years' observation to include the latent period.

Cruickshank and Gourevitch[24] have presented 87 cases of cancer of the hands and forearms seen by them in a ten-year period. By study of their job exposures they conclude that these men suffered epithelioma as a result of exposure to various oils in their occupation as "metal workers." These authors point out that many such cancers appear after the worker has retired so that the job connection is missed. Age at time of onset, rate of incidence and case mortality show these skin cancers to behave very much like occupational scrotal cancer, which is also due to certain oils.

By clinical and animal experimental study, Cruickshank and Squire,[25] in 1950, demonstrated a significant incidence of scrotal cancer due to a cutting oil used in a machine shop. Thirty-four cases were studied, occurring in a ten-year period — 6 in machine operation, 6 in workers exposed to oil in the engineering industry, and 13 after notable exposure to tar and pitch.

Snegireff and Lombard[26] have discussed arsenic as an occupational cause of cancer, with comments on the controversial evidence in the literature. Field visits were made to certain plants using arsenic in the metallurgical industries. By studying cancer death rates in these plants and in plants where no arsenic was handled, the authors conclude that factors in addition to arsenic trioxide must be responsible for the cancer deaths.

Hueper,[27] in an exhaustive study of, as he puts it, environmental lung cancer, brings out current and past evidence in the literature to support the contention that there is a true increase of this disease. Statistical evidence is marshaled to show this increase, independent of age composition of the population and improved diagnosis and pathological study. He demonstrates that when environmental factors are ironed out the sex difference vanishes. The great interest in cigarette smoking as a cause of lung cancer in men, as urged by Graham[28] and Wynder and Graham,[29] is certainly an important part of this discussion. It may be that cigarette smoking in addition to an occupational insult can be shown as a significant cause of malignant lesions of the lung.

The subject of cancer of the lung in significant incidence among workers in the chromate-manufacturing industry has received considerable attention recently here and abroad. Mancuso,[30] in a complete study in Ohio, found that there were fifteen times as many deaths from lung cancer among chromate workers as in the general population of the county in which the plant is located. The period before onset was ten and six-tenths years. Mancuso thought his evidence pointed toward the insoluble chromium compounds as cause. He found, by blood and urine assay of workers exposed to chromium, that the chemical is retained in the body for several years after cessation of exposure, which Mancuso suggests may have direct bearing on the carcinogenic effect of this chemical. Baetjer,[31] in 1950, made a satisfactory study of hospital records of two Baltimore hospitals from which she concluded that during the previous twenty years the number of deaths due to cancer of lungs and bronchi was greater among workers from the chromate-producing industry than in the general population. At the same time Baetjer reviewed the available data and concluded that there is good evidence of increase in the incidence of respiratory-tract cancer in German and American chromate-manufacturing plants. It must be made clear that chromate-finished materials as such have not been incriminated as carcinogenic.

Bidstrup,[32] in a preliminary report, finds only 1 case of pulmonary cancer in 724 workers in the British chromate industry. There is no explanation at present for this discrepancy between the English and German and American experience. Buckell and Harvey[33] find chromium exposures higher in the three plants in Great Britain than those reported from a factory in the United States of America.

Bidstrup,[34] discussing the increase in pulmonary cancer, describes the British Mond process of extracting nickel. She reports that in four years 20 cases of cancer of the ethmoid and 19 cases of lung cancer were officially reported, though others were known to have occurred. Both nickel and arsenic have been designated as causative. No case of cancer caused by nickel has been described in America.

Asbestos as a cause of respiratory-tract carcinoma is disputed by Vorwald[35] in this country. English reports seem convincing, especially that of Merewether in the "Annual Report of Chief Inspector of Factories," published in London in 1947, presenting 235 cases of asbestosis with 13.2 per cent incidence of pulmonary cancer (compared with 1.0 per cent in the general adult population). Stoll and his associates[36] presented a case in 1951 of a man exposed for four and one-half years as a shipyard worker using asbestos. He neglected to wear the mask provided. About five years after this and after a period of cough, dyspnea and weight loss, the patient died. Asbestos bodies were seen throughout the lung. A bronchogenic carcinoma was found, with metastases to kidneys, brain and liver. A striking case of pulmonary adenocarcinoma was reported by Isselbacker et al.[36a] in 1952 in a man who worked for seven years sorting and carding asbestos fibers intended for insulation manufacturing. Dangerous levels of asbestos in his working environment were documented. Five years after leaving this work the patient went rapidly downhill with backache, dyspnea and weight loss as presenting symptoms. At autopsy extensive pulmonary adenocarcinoma with metastases to the vertebras was found. These cases have particular significance since there are nearly 10,000 workers exposed to varying amounts of asbestos in American industry, many of them in some operations not properly safeguarded against a potential carcinogen.

Cromwell and Papanicolaou[37] urge the taking of routine sputum smears from workers exposed to recognized respiratory-tract carcinogens. They point out that such sputums must be examined by a well-trained person, a procedure that can now be attained at reasonable cost.

Scott[38] reports 66 cases of tumor of the bladder in workers in an English dyestuff factory. The Italian workers, Barsotti and Vigliani,[39] by insistence on cystoscopy routinely in dyestuff workers, report 18 carcinomas and 11 papillomas with-

out symptoms. Benzidine and beta-naphthylamine by both reports have the greatest carcinogenicity. Truhaut[40] reports recent similar experience in French industries with the aromatic amines required in dyestuff manufacture. Cromwell and Papanicolaou[37] point out that since cystoscopy is routine in certain plants it would be worth while examining the urine for cancer cells as a reliable guide to very early bladder tumors.

Medical Aspects of Radiation

Knowledge of disease produced by the physical insults of various kinds and quantities of radiant energy is a requirement of modern medicine. The expansion of atomic-energy development, the use of radiography in industry, diagnostic and therapeutic radiation, the increase in research in agriculture, and the increased use of radioactive isotopes in medicine and industry mean that more and more persons may be exposed to potentially harmful effect in the absence of intelligent protection.

Workers in atomic-energy development are remarkably well protected owing to excellent technical skills made available by industrial hygienists and physicists with special training who are variously termed radiologic safety officers or health physicists. Eisenbud,[41] in a recent article, discusses the problems involved in such protection. He reports that between 1943 and 1950 there have been but 2 deaths and 12 injuries directly due to radiation exposure received by workers engaged in this great government enterprise.

An interesting, well illustrated symposium on so-called radiologic health held at the University of Michigan School of Public Health was published in April, 1951.[42] Anyone wishing to visualize the practical detail of worker protection by monitoring, shielding and distance and the public-health protection of safe disposal of radioactive wastes would do well to read this material. Hempelmann, Lisco and Hoffman[43] in an article published in 1952, described in great detail the accidental deaths and injuries resulting from the work of scientists on atomic-bomb physics. These descriptions of acute radiation reaction in varying degree should be read as background for any possible civilian disaster consequent on military use of atomic energy. Hempelmann[44] recently presented the clinical data on these disasters in the *Journal*.

The possibility of delayed effect of low-level doses of body penetrating radiation received over a period of years by workers in atomic-energy development has been carefully considered. From earlier knowledge of radiation damage, blood dyscrasias are most likely to occur. As a result, carefully obtained complete blood counts taken before radiation and during exposure at varying intervals depending on the character of exposure are the most widely used tool for medical control of exposed workers. After periods of up to seven years of taking such counts, many observers believe that this procedure is expensive, wastes time and is often misleading because of the many variables in the technic of counting, the physiologic variations and low-grade infections influencing the hemogram. From a practical standpoint, a pre-exposure and an annual count of all personnel exposed to permissible doses of radiation is probably sufficient for medicolegal protection. Chamberlain, Turner and Williams,[45] working at Harwell, the British Atomic Energy Establishment, in a critical review of the literature and their own data, find little protective comfort in the three-monthly and six-monthly routine counts currently used. They find that over a period of months a worker's count may fall 30 per cent in 10 per cent of successive counts taken at random. Their experience leads them to plan for more frequent blood counts on workers receiving "appreciable radiation regularly" with a comparable number of controls. Knowlton [46] reported on 10 atomic-energy laboratory workers receiving 0.2 r of gamma radiation for seventy-seven consecutive weeks who showed a slight but statistically valid fall in total white-cell, absolute neutrophil and lymphocyte counts. These same workers showed a gradual return to normal levels during six months away from radiation. This return to normal suggests the validity of the current belief that such a reversible effect of radiation is probably benign. Dickie and Hempelmann,[47] in a complete study of the blood smears of 364 exposed and unexposed workers at an atomic-energy laboratory, described an increase in intracellular bodies (called Dickie bodies) in lymphocytes in supravital stained preparations of exposed workers. While not specific, this is evidence of an effect of radiation that may or may not prove ultimately to be pathologically important. Bilobed nuclei in the peripheral blood smears of cyclotron workers were reported first by Ingram and Barnes[48] at Rochester and later by Carter at Los Alamos, and Browning in England. We have also seen these cells in Massachusetts Institute of Technology cyclotron workers. The cells appear to be large lymphocytes in configuration and reaction to Wright's stain. The cytoplasm is clear, light blue and sometimes appears "clumpy" with peroxidase stain, to which the cells are negative. Such cells have been produced in rabbits and dogs by the use of x-rays and with the dogs placed near a cyclotron. Ingram and Barnes suggest that this may be a means of identifying potentially harmful exposures.

Atomic-energy projects must use uranium in many forms, plutonium and other naturally and artificially radioactive materials. These laboratories have research groups constantly studying biologic effects of such exposures to the end that the workers may be protected.

In 1951 a volume edited by R. S. Stone,[49] entitled *Industrial Medicine on the Plutonium Project*,

was published as part of the National Nuclear Energy Series. Here are collected valuable and well-arranged data compiled while Stone and his associates were devising medical control for the hazards associated with wartime atomic-energy development. Studies of hematology, biochemistry and heavy radioactive metal excretion are presented and discussed, providing excellent background knowledge.

The most important matters for the physician to grasp are the basic facts of the quality and quantity of radiant energy received by the worker-patient whose clinical status he may wish to evaluate. With this knowledge, it is then imperative to know whether the material was ingested or inhaled, to determine its possible action. Furthermore, the result of radioactive insult may depend on body penetration with possible bone-marrow effect or local action on the body surface. It must be known whether the action took place over a localized area or whether it involved the whole body. Most of the literature in this field is too vast for useful review. The interested reader is referred to the Nuclear Science Abstracts published by the Technical Information Service of the Atomic Energy Commission at Oak Ridge to be found now in most medical libraries, and to current publications such as *The Effects of Atomic Weapons*[50] and *Atomic Medicine*.[51]

Hamilton,[52] at Berkeley, Langham,[53] at Los Alamos, and Lisco, Finkel and Brues[54] at the Argonne Laboratory, have done extensive work showing the carcinogenicity and bone-seeking properties of the fission products and heavy radioactive elements that must be used in atomic-energy development. Hodge[55] has presented an interesting summary of knowledge of the mechanism of uranium poisoning acquired in the extensive studies done during and since World War II. It has been learned that some of the uranium is deposited in bone "through exchange with calcium ions at the surface of the bone mineral." The author says that although most of the uranium is excreted in the urine, some of the element damages the living cells of the renal tubules by reason of local injury.

Hodge, Maynard and Downs[56] showed that in small animals polyphosphates administered in single and multiple injections have markedly reduced the mortality of acute experimental uranium poisoning. Thus far these materials have not been incriminated as causing job-related disease. Methods of assay of urine[53] and workroom air for quantities of hazardous material have been devised to ensure protection. If it is found by such a study that a worker carries an amount of a radioactive material in his body more than that considered safe, he is removed permanently from work involving radiation exposure.

Schubert[57] continues his study of methods for removing heavy radioactive material from the body skeleton. He finds zirconium the most useful, reporting that adsorption of the radioelement on colloidal aggregates of zirconium is the probable mechanism of action. Foreman[58] has been studying the use of ethylene diamine tetra-acetic acid, a chelating agent, in, he believes, successful attempts to remove heavy radioactive material from the body.

Because present permissible levels of worker exposure to radiation depend on their findings, the cases of radium poisoning reported by Martland[59] and Evans[60] are being restudied. Owing to the delayed effect of radium deposited in the body "new" cases are currently being studied. Evans, with more sensitive devices, is exploring Hempelmann's revival of Martland's early observation that the deaths of the dial painters were largely due to the mesothorium content of the luminizing solution. Aub et al.[61] are presenting these data for current publication. The Argonne workers[62] are studying a group of patients who received radium therapeutically by mouth and vein. The patients in this group have survived longer with less disease. These studies are of the greatest importance in determining whether the permissible level of 0.3 r per week is safe as a lifetime worker dose of radiation. All other values for whole-body radiation presently used stem from this value, which has been reduced from the figure of 0.1 r per day derived from Evans'[60] study of the casualties among the New Jersey radium-dial painters presented by Martland[59] in 1925. The Atomic Energy Commission has bound together (1951) and reprinted all Martland's papers on the subject of radium poisoning, an invaluable collection* for those interested in understanding potential hazards in work with radioactive materials.

Hunter et al.[63] have presented a careful study of radiation received in diagnostic and therapeutic work at the Massachusetts General Hospital. Diagnostic workers received below 0.1 r in two weeks, and radium workers less than 0.3 r in two weeks. The blood counts showed no changes. Past or even present poor practice, however, has resulted in continued reports of radiation damage among professional people. Daland,[64] in 1951, reported injuries among surgeons because of prolonged use of the fluoroscope for fracture manipulation. Peller and Pick,[65] in a letter to the *Journal of the American Medical Association* in October, 1951, presented up-to-date statistics to show the relatively high incidence of leukemia among radiologists. Muth and Roth[66] described the case of a chemist, working without proper protection with radium and mesothorium. In 1940 signs of pulmonary damage appeared with rales and x-ray evidence of "upper field striae and patches of a bronchopneumonic character." In 1943 an anemia began. Postmortem study after death in 1948 revealed pulmonary fibrosis and aplastic anemia. The con-

*This may be obtained at The Technical Information Service, Atomic Energy Commission, Oak Ridge, Tennessee.

tent of radioactive material in this man's body was estimated as 3.5 microgm.

In industry static eliminators[67] made of radium or polonium are used to prevent fires from static electricity and make easier handling of goods in textile and paper production. No reports of radiation injury have as yet come to light, though it is known that many of these eliminators were so carelessly made as to allow small amounts of body-penetrating radiation to reach the worker. Some industries do not find this equipment as useful as advertised. There are no current reports of injury from industrial radiography employed for metal parts, golf-ball and candy inspection. Bourne and Cordier,[68] in an Ohio survey, found 69 industrial x-ray units in use. They report that there may be a hazard because of the high energy used and that since only 2 of 42 plants had had a radiation survey, it seems true that "users of the apparatus have not kept pace with our knowledge of radiation." Shoe-fitting fluoroscopy has received national publicity and some restrictive legislation. Radiation injury prophesied has not been reported, perhaps, as in other radiation exposures, because of the delayed effect of this physical insult.

In 1950 Cogan[69] reviewed completely the effect of ultraviolet and infrared light, as well as various radioactive energies, on the eyes, a potential worker problem in many operations. He pointed out that the biologic effect of the various forms of radiant energy depends on their power of penetration and absorption. For instance, only 10 per cent of the violet rays of the spectrum can reach the retina. Cataracts have been reported in a number of survivors of the Hiroshima bombing[70] and in a few workers exposed at cyclotrons and in atomic-energy laboratories to fairly heavy doses of penetrating radiation. Gamma rays and fast neutrons as well as x-rays are considered cataractogenic. Cogan and Donaldson[71] regarded the dose necessary to produce a cataract as between 500 and 1000 r of x-rays of 100 kv. energy or more. This figure has been developed from experimental production of radiation cataracts.

Diseases of the Respiratory Tract of Occupational Origin

Pulmonary diseases of occupational origin have received, as they deserve by incidence and interest, considerable recent attention. For clarity in this report it is repeated that pneumoconiosis means dust in the lungs to which there may or may not be reaction. One usually classifies the pneumoconioses by specifying the etiology in the diagnosis as siderosis, referring to iron-produced pneumoconiosis, and silicosis, as that produced by SiO_2, silicon dioxide, familiar as quartz. Physicians cannot remind themselves too often that exact exposure histories must be taken in the establishment of a diagnosis of job-related pulmonary disease. Inhalation of sufficient quantity of a material that will cause a pathologic reaction, of a particle size that can reach the lungs, must be described, with, of course, allowance for variation in individual response.

B. M. Wright,[72] discussing the weaknesses of the theory that silicosis is caused by "freshly fractured surfaces," brings out many interesting points in the exact study of the etiology of silicosis and other dust diseases. He points out that even if the harmful silica particles have fresh surfaces on inhalation, biologic fluids would soon destroy any activity dependent on such surfaces. Wright urges a conscientious study of the dust cloud as it actually exists in the silicotic person's working atmosphere. Ray, King and Harrison[73] have been able to demonstrate convincingly in animal studies that the pathologic response of the lungs to quartz varies with the amount. At 2-mg. dosages no lung fibrosis was found; at 5 and 10 mg. nodules were found; at 30 mg. collagenous nodules were produced; and at 75-mg. doses the nodules were collagenous and confluent. The same workers showed that the presence of coal or graphite enhances the pathogenic effects of silica. Bedford and Warner[74] studied the size distribution of dust particles from the lung of a shot-firer who died of silicosis after many years' work in an anthracite mine. They showed a remarkably close correlation between the size distribution in the airborne dust where the man worked and in his lung. Changes in industrial operations and the introduction of previously unused respirable materials mean that "new" pneumoconioses will be reported.

Disease-Producing Pneumoconiosis

Dreesen,[75] in a report published in 1949, says that there are an estimated 110,000 persons in the United States of America with x-ray-demonstrable silicosis out of 1,000,000 workers exposed to silica, and of this number 4000 to 5000 had Stage III silicosis at the time of the National Silicosis Conference of 1936. He reports that if all mineral dusts are included the number is doubled. There were 490 deaths reported in this country in 1946 from pneumoconioses per se. Occupational disease is not reportable in this country, but figures from urban hospitals, Dreesen continues, such as those in Los Angeles and Detroit, suggest that 0.5 to 3 per cent of all patients have silicosis. This leads to the estimate of 500,000 to 1,000,000 pneumoconiotic workers from the 50,000,000 to 60,000,000 workers in industry. Mining, stone cutting, pottery manufacture and foundry work continue to be the chief sources of cases of disabling dust disease of the lung.

Since complicating acid-fast infection is the chief source of disability and death in silicosis, the article of Vorwald et al[76] is important as showing that in animal experiments vaccination with BCG does not prevent and retard animal tuberculo-

silicosis. Treatment of silicosis with aluminum dust has not been endorsed by the American Medical Association after a survey of the present clinical evidence by Brown and Van Winkle.[77] It is pointed out that there is subjective but no objective evidence of improvement in the lung function of silicotic patients after aluminum therapy. Although no evidence that aluminum is harmful to the normal or silicotic lung was found, these authors reported that certain Canadian miners showed progression of x-ray changes in spite of the therapy. Brown and Van Winkle believe that the study required to evaluate aluminum prophylactic therapy cannot be undertaken by industry but should be left to the Saranac Laboratory, which is now started on an investigation. Evans and Schweisheimer,[78] writing from South Africa in 1950, state that the inhalation of aluminum is helpful for the prevention and treatment of silicosis, but no supporting evidence is given. Meanwhile, reports of aluminosis of the lung continued to appear. Undoubtedly, the doses used in aluminum therapy are very different, but the fact of dust disease from aluminum causes persistent comment. Mödder and Schmitt[79] report "heavy damage" to the lungs of 18 operators of the smelting and alloying department of an aluminum smelting plant. Ivanova and Ostrovskaja[80] found evidence in the literature, including clinical and animal experimental data, that aluminum dust even in small amounts is far from harmless. They then gave aluminum powder to small animals and found nodules of fibrous tissue in the terminal bronchioles three months after intratracheal administration. On the other hand, King and his associates[81] discovered that if they dusted rats with quartz and aluminum there was "almost no reticulinosis at 200 days and only very slight reticulinosis at 300 days." There appears to be much more animal work and human observation required to resolve the question of the usefulness of aluminum therapy in silicosis.

While one is considering the therapy of silicosis, it is interesting to note that Kennedy et al.[82] report some improvement measured by pulmonary-function tests in the treatment of silicosis with ACTH. Many more data will be needed to evaluate this single observation.

Meyer and Solomon[83] found a 12.8 per cent incidence of silicosis in their survey of an iron foundry. They made the interesting observation during this study that silicon dioxide could be demonstrated in the sputum in 4 cases of severe silicosis by x-ray diffraction analysis, whereas none was found in 10 control patients suffering from a variety of lung diseases. This observation might be helpful in diagnostic problems after more work has been done, as the authors urge, if x-ray diffraction facilities become more widely available.

A great deal of excellent work has been done in recent years to increase knowledge of the pulmonary disease suffered by coal workers. British workers use the term "coal worker's pneumoconiosis," which impresses me as ideal. In general, American terminology prefers anthracosilicosis as the accepted designation. Meiklejohn[84, 85] has recently written a history of coal worker's lung disease in Great Britain (1800–1920), showing the rise and fall of interest in as well as knowledge of this field as industry expands. McCallum[86] reports well marked chest x-ray evidence of pneumoconiosis in between 3 and 6 per cent of the coal workers in Northeastern England. These changes appear similar to those found in the soft-coal miners of South Wales. Cochrane and his associates[87] at the Pneumoconiosis Research Center in Cardiff, Wales, have presented evidence that if workers are removed from harmful dust when early chest x-ray changes appear they will not have disabling disease. They suggest periodic examinations to implement such a program. Difficulties of compensation, separation of the man from his work and creation of a scare that would keep workers from applying are not, they believe insurmountable obstacles. Cochrane et al. correctly point out that ventilation control of an excess of harmful dust must be pushed and that long-term periodic examination of workers will be a check on the effectiveness of engineering measures. Meiklejohn[88] has questioned the value of such periodic examinations because of the difficulties of relying on radiographic changes, which are subject to technical variation and reader error. As he says, the workman who feels well in the early stages of an x-ray pneumoconiosis may be unwilling to leave his job. In America observers agree with Meiklejohn that lowering of exposure after changes in operation and failures in engineering control probably means that the real control lies with the measures that will prevent the workman from inhaling harmful dusts. Corcoran[89] provided a discussion of the x-ray pattern of anthracosilicosis, or coal worker's pneumoconiosis, as seen in American miners. He described uniformly distributed nodules as characteristic, with later coalescence. This author states that in his experience five to seven years elapse after harmful dust exposure before x-ray changes are seen.

Fletcher and Oldham[90] have made progress toward eliminating the errors involved in the use of chest x-ray examinations to detect early evidence of coal worker's pneumoconiosis. They suggest that standard films be used for comparison and that at least two observers read the films on two separate occasions. They have tried further to lessen error by dividing the changes into five categories, which they define carefully. These groups range from 0, meaning normal limits, through minimal simple and moderate simple pneumoconiosis to marked and maximal simple pneumoconiosis. These efforts seem well worth while in view of the damage done by faulty x-ray interpretation. Cochrane, Davies and

Fletcher,[91] of the Pneumoconiosis Research Unit, have recommended attempts at international reading of chest x-ray films, which they call "Entente Radiologique." A large number of films were read in consultation by British and French physicians, and a joint classification was proposed. This classification is now on trial in various centers in France, Belgium and Holland. A similar enterprise has been reported by Garland and Cochrane,[92] the former in San Francisco, and the latter in Cardiff, Wales. Eighty-nine chest x-ray films of Welsh coal workers were independently read twice by each of 6 British and 3 American observers. Because of the percentage of disagreement, the authors conclude that real observer error exists in more than one country and propose that more effort be made toward correction. Gough and his associates,[93] by comparing autopsy lung sections of 76 coal workers in South Wales, add to the evidence that chest x-ray examinations are only a part of a diagnostic whole in the evaluation of pneumoconiosis. They found fairly good correlation in moderate or severe pneumoconiosis between x-ray signs and disease, but poor parallel in early pneumoconiosis producing widespread focal emphysema. This lack of relation is important when one is judging the usefulness of repeated routine chest x-ray studies in groups of workers exposed to harmful dusts. Gough et al. showed that large focal shadows of infected pneumoconiosis were occasionally mistaken for carcinoma of the bronchus.

An extremely important contribution to the field of diagnosis of job-related pulmonary disease has been made by Motley and his co-workers[94] during recent years of work carried on in the Cardio-Respiratory Laboratory of the Jefferson Medical College Hospital. By clinical investigation of men engaged in mining bituminous and anthracite coal, Motley and his group[95] have developed the pulmonary-function studies that are of value in diagnosis of coal worker's pneumoconiosis, called anthracosilicosis in America. After study of 56 bituminous and 216 anthracite miners[96] they conclude that the pulmonary-function disability is similar in the two groups and that fibrosis resulting from dust inhalation results in unequal alveolar aeration and perfusion, with consequent decrease in the flow of oxygen to the pulmonary arterial bed. In about two thirds of their cases there was a significant degree of emphysema, which, as it increased, was found to be significantly correlated with evidence of strain on the right side of the heart. Motley points out the poor correlation among x-ray findings, subjective complaints and changes in measurements of pulmonary function. As might have been expected because of individual variations, no one determination was found to be satisfactory, and Motley et al. believe that measurements of ventilation, residual air, oxygen uptake from the air breathed, oxygen saturation of arterial blood and pulmonary circulation are necessary before a complete opinion of the respiratory ability of a patient with pneumoconiosis can be reached.

Slesinger[97] has presented his observations of anthracosilicosis, or coal worker's pneumoconiosis, among soft coal workers. He agrees with Gough[98] that chronic cor pulmonale is a common cause of disability in this disease. Gough, in his pathological studies, has shown that the widespread fibrosis of coal worker's pneumoconiosis leads to changes in the pulmonary circulation. Slesinger[97] cites Lang as finding 24 per cent of 148 coal workers with electrocardiographic evidence of strain of the right side of the heart. This report does not always keep clear the distinction between silicosis (pure SiO_2 exposure), with its clinical and pathological pattern, and anthracosilicosis (exposure to carbon, an inert dust, combined with SiO_2), with its peculiar characteristics.

Heppleston,[99] working with Gough, and Gough independently[98] have, after study of 700 autopsies, written with authority of the pathogenesis of coal worker's pneumoconiosis. They describe the disease as occurring in two forms: a benign or simple pneumoconiosis of coal workers in which dust deposition has resulted in widespread focal emphysema; and an infective pneumoconiosis of coal workers in which massive fibrosis is superimposed on simple pneumoconiosis as the result of infection developing in a dust-laden lung. Heppleston says that the pneumoconiosis of Welsh, American and Scotch coal workers is identical. His studies lead him to believe that the infection is probably tuberculous although the organism is found in only about 65 per cent of the cases. This study clarifies the differential points between silicosis and coal worker's pneumoconiosis and makes intelligible their clinical course, x-ray picture and findings of pulmonary-function study.

Gordon and Motley,[100, 101] understanding the physiologic changes involved in silicosis and coal worker's pneumoconiosis, have devised some worthwhile forms of treatment. Because of a large element of bronchospasm, bronchodilators are prescribed as an aerosol to facilitate an increase in the air moving in and out of the alveoli. Intermittent positive-pressure oxygen is used for short periods, with remarkable subjective improvement. Pulmonary-function studies show that a real change has occurred. Gordon and Motley report that such therapy in 500 cases of coal worker's pneumoconiosis resulted in expectoration of thick, tenacious "black" sputum in 50 per cent of the cases, with great relief of dyspnea and the asthma-like attacks. Physical signs altered with increase in aeration and drainage, but no x-ray changes as a result of treatment were detected.

Asbestos has been discussed as a potential carcinogen. Vorwald and his co-workers[102] at the Saranac Laboratory presented (1951) the results of their experimental studies of asbestosis. They pointed out that the term asbestos is applied to a

variety of minerals, which are silicates. The Saranac group found that long rather than short respirable fibers produce the peribronchiolar fibrosis known to be the disabling pathologic feature in asbestosis. Earlier work was confirmed by the finding that the mode of action is mechanical and, as might have been expected, depends on length and intensity of exposure. Experimental asbestosis ceases to progress when exposure stops. In worker asbestosis the disease produced if the exposure has been sufficient leads to focal atelectasis with superimposed infection and resultant chronic pulmonary disability. The asbestos bodies, Vorwald's group finds, are the result of "coating of the fibers by blood and tissue elements which results in loss of ability of the fiber to produce fibrosis." Experimental tuberculosis was not altered by the inhalation of asbestos fibers.

Experience* with 1 case of asbestosis treated with ACTH showed no improvement as measured by studies of pulmonary function before and after treatment.

Shaver[102] continues to report on the pulmonary disease associated with exposure to fine bauxite fume, largely aluminum oxide, when this material is used in the manufacture of abrasives. Shaver reports 34 well established and 38 early cases. In reviewing his experience he concludes that the disease went unrecognized before World War II and that some of the early cases now occurring may be related to heavy fume concentration of wartime. Shaver finds that early removal from exposure results in stabilizing the process without serious disability. Once established, the disease is usually of rapid progression, with extreme dyspnea. Acute infection appears to play no part in the course of this disease.

Talc is hydrated magnesium silicate and whether or not its use occupationally results in a disabling pneumoconiosis continues to be a subject of discussion. Hogue and Mallette[104] point out that there are many commercial applications of "talc." The materials used may be quite different in mineralogic and chemical character. They report that physical examinations and chest x-ray studies of 20 men exposed to talc in the rubber-tire industry showed no changes considered job-related after ten to thirty-six years' exposure. They urge careful definition of chemical and mineralogic composition in the description of disabling pneumoconiosis considered to be due to inhalation of silicate minerals. McLaughlin, Rogers and Dunham[105] present evidence that moderately advanced pneumoconiosis was caused by talc in the lungs of a man who had worked thirty-seven years in a rubber-tire factory. X-ray diffraction studies of the lung revealed the presence of talc. Jaques and Benirschke[106] have recently described the finding of talcosis, as they term it, in the lung, stomach and heart of a forty-two-year-old man who was exposed to talcum powder in a patent-leather shoe factory for six years. Talc crystals were identified in the pathologic lesions. McLaughlin[107] and Jaques and Benirschke have reviewed the literature. There does appear to be evidence to justify the conclusion that at a certain level and duration of exposure to properly identified talc, disabling pneumoconiosis may result in certain cases. Since industry uses many dusting compounds, the warning of Hogue and Mallette[104] that the term "talc" is insufficient in correct diagnosis of unclassified pneumoconiosis should be heeded.

Troisi[108] reports that silicate dust inhalation occurring in the glazing of iron parts of cookers has caused a true lung fibrosis demonstrable by x-ray study. The 4 workers whose cases are reported showed no disability. This may prove to be a benign pneumoconiosis.

Dunner and Bagnall[109] believe that local deposits of graphite in the lung produce fibrosis, with gradual loss of blood supply, local necrosis and gradual contraction of the fibrous tissue, leading to cyst formation. They report on their observations of workmen in a factory producing "black lead." A variety of x-ray changes including a "snow-storm" pattern are reported. Dunner and Bagnall say that in a few cases only one lung was involved. The authors believe that there is no relation between duration of exposure and date or extent of lesions. Harding and Oliver[110] have also presented evidence of a graphite pneumoconiosis. Jaffé[111] reported the case of a man who worked for five years in an intense exposure to dust in a graphite mine. Twenty years later a lung tumor removed showed granulomatous reaction, with fibrosis, necrosis and cavitation, as well as obliterative changes in blood vessels and bronchi. There were giant cells and many pathological features similar to those described by Harding and Oliver. Jaffé discusses the role of silica in producing such a reaction. In his case the content of the lung silica was the same as that of normal controls, and the graphite content was high. Parmeggiani[112] carried out a clinical and x-ray study among graphite workers. Among 415 workers examined he found 24 cases of pneumoconiosis, which, he believes, should be termed a benign mixed pneumoconiosis since the graphite contains 10 per cent free silica. Dyspnea is the chief symptom; x-ray therapy changes the finding. Emphysema is rare, and the incidence of tuberculosis less frequent than in silicosis, according to Parmeggiani. McCord[113] reviewed the available evidence in 1949 and concluded that disability from graphite has not been proved. One suspects that this may be failure of the literature. H. E. Harding† and T. E. MacMahan‡ have shown me autopsied lungs of graphite workers who had died of cor pulmonale in which the mechan-

*Unpublished data of the Occupational Medical Clinic, Massachusetts General Hospital.

†Personal communication, 1950.
‡Personal communication, 1947.

ical effect of the dust was dramatically demonstrated.

Diatomaceous earth used in insulation and sugar refining has been shown to produce a rapid fibrotic reaction in workers inhaling the calcined dust. Vorwald et al.[114] report that this material as a mineral is essentially pure silica but that commercially it contains impurities that are chiefly clay. Experimental and human pneumoconiosis from diatomaceous earth has an unfavorable effect on associated tuberculosis. Animal studies show a widespread pulmonary fibrosis as the important lesion. Vigliani,[115] reporting clinical experience, described a rapidly developing dyspnea, with frequent occurrence of spontaneous pneumothorax. Chest x-ray study showed fine mottling throughout and, in advanced cases, "fluffy or massive shadows." Autopsy showed granulomas, marked emphysema and pleural adhesions.

Harding and Massie[116] have presented 8 cases of pneumoconiosis in men engaged for long periods as boiler scalers in shipyards and large industrial plants. The reaction pathologically was shown to be similar to the coal worker's pneumoconiosis. These men were exposed to large amounts of dust of varying concentrations of carbon, silica and iron oxide. Harding and Massie refer to this pulmonary disease as a mixed dust fibrosis and believe it is a modified form of silicosis. Respiratory infection in workers with this type of pneumoconiosis caused disability.

(To be concluded)

References

1. Wolff, A. H., and Heimann, H. Industrial anthrax in United States: epidemiologic study. *Am. J. Hyg.* **53**:80-107, 1951.
2. Kolb, R. W., et al. Disinfective action of methyl bromide, methanol, and hydrogen bromide on anthrax spores. *Arch. Indust. Hyg.* **5**:354-364, 1952.
3. Editorial. Anthrax. *New Eng. J. Med.* **236**:882, 1947.
4. Clarke, P. S. Chloramphenicol in treatment of cutaneous anthrax. *Brit. M. J.* 1:86, 1952.
5. Gold, H., and Boger, W. P. Newer antibiotics in treatment of anthrax. *New Eng. J. Med.* **244**:391-394, 1951.
6. Abrams, H. K., and Warr, P. Occupational diseases transmitted via contact with animals and animal products. *Indust. Med.* **20**:341-351, 1951.
7. Bell, J. A., Beck, M. D., and Huebner, R. J. Epidemiologic studies of Q fever in Southern California. *J. A. M. A.* **142**:868-872, 1950.
8. Sigel, M. M., et al. Q fever in wool and hair processing plant. *Am. J. Pub. Health* **40**:524-532, 1950.
9. Skinner, J. B., and Maloof, C. C. Medical hazards associated with fish industry in Massachusetts. *New Eng. J. Med.* **240**:256, 1949.
10. Skinner, J. B. Newer occupational disease. *Indust. Med.* **20**:133, 1951.
11. Tulipan, L., and Muskatblit, E. Industrial sporotrichosis. *Compens. Med.* **3**:36-38, 1950.
12. Sulkin, S. E., and Pike, R. M. Survey of laboratory-acquired infections. *Am. J. Pub. Health* **41**:769-781, 1951.
13. Editorial. Tularemia prophylaxis. *J. A. M. A.* **144**:1468, 1950.
14. Lepine, P., Atanasiu, P., and Gareau, G. Infection of laboratory workers by virus of Newcastle disease: three cases. *Ann. Inst. Pasteur* **79**:193-196, 1950.
15. Shaw, E. W., Melnick, J. L., and Curnen, E. C. Infection of laboratory workers with Coxsackie viruses. *Ann. Int. Med.* **33**:32-40, 1950.
16. Terry, L. L., Lewis, J. L., Jr., and Sessoms, S. M. Laboratory infection with Leishmania donovani: case report. *Am. J. Trop. Med.* **30**:643-649, 1950.
17. Hueper, W. C. Environmental cancer hazards caused by industrial air pollution; introductory comment to discussion. *Arch. Indust. Hyg.* **2**:324-328, 1950.
18. *Idem.* Occupational cancer hazards in American industries. *Arch. Indust. Hyg.* **5**:201-203, 1952.
19. Mayers, M. R. Program of New York State Occupational cancer committee. *Arch. Indust. Hyg.* **5**:279-283, 1952.
20. Merrill, E. D. New Jersey occupational cancer survey. *Arch. Indust. Hyg.* **5**:284-287, 1952.
21. Heller, J. R. Chemical carcinogens. *Arch. Indust. Hyg.* **2**:390-399, 1950.
22. Symposium on cancer control program for high-boiling catalytically cracked oils. *Arch. Indust. Hyg.* **4**:297-345, 1951.
23. Ingalls, T. H. Incidence of cancer in carbon black industry. *Arch. Indust. Hyg.* **1**:662-676, 1950.
24. Cruickshank, C. N. D., and Gourevitch, A. Skin cancer of hand and forearm. *Brit. J. Indust. Med.* **9**:74-79, 1952.
25. Cruickshank, C. N. D., and Squire, J. R. Skin cancer in engineering industry from use of mineral oil. *Brit. J. Indust. Med.* **7**:1-11, 1950.
26. Snegireff, L. S., and Lombard, O. M. Arsenic and cancer: observations in metallurgical industry. *Arch. Indust. Hyg.* **4**:199-205, 1951.
27. Hueper, W. C. Environmental lung cancer. *Indust. Med.* **20**:49-62, 1951.
28. Graham, E. A. Some questions about bronchiogenic carcinoma. *New Eng. J. Med.* **245**:389-396, 1951.
29. Wynder, E. L., and Graham, E. A. Etiologic factors in bronchiogenic carcinoma, with special reference to industrial exposures. *Arch. Indust. Hyg.* **4**:221-235, 1951.
30. Mancuso, T. F., and Huepner, W. C. Occupational cancer and other health hazards in chromate plant: medical appraisal. I. Lung cancers in chromate workers. II. Clinical and toxicologic aspects. *Indust. Med.* **20**:358-363 and 393-407, 1951.
31. Baetjer, A. M. Pulmonary carcinoma in chromate workers: review of literature and report of cases. II. Incidence on basis of hospital records. *Arch. Indust. Hyg.* **2**:487-504 and 505-516, 1950.
32. Bidstrup, P. L. Carcinoma of lung in chromate workers. *Brit. J. Indust. Med.* **8**:302-305, 1951.
33. Buckell, M., and Harvey, D. G., Environmental study of chromate industry. *Brit. J. Indust. Med.* **8**:298-301, 1951.
34. Bidstrup, P. L. Cancer of lung in nickel, arsenic, and chromate workers. *Arch. belges med. sociale et d'hyg.* **8**:500-505, 1950. (*Arch. Indust. Hyg.* **5**:403, 1952. Abstr.)
35. Vorwald, A. J. Personal communication.
36. Stoll, R., Bass, R., and Angrist, A. A. Asbestosis associated with bronchogenic carcinoma. *Arch. Int. Med.* **88**:831-834, 1951.

36a. Isselbacker, K., Klaus, H., and Hardy, H. L. Unpublished data.

37. Cromwell, H. A., and Papanicolaou, G. N. Use of cytologic method in industrial medicine, with special reference to tumors of lung and bladder. *Arch. Indust. Hyg.* **5**:232, 1952.
38. Scott, T. S. Incidence of bladder tumours in dyestuffs factory. *Brit. J. Indust. Med.* **9**:127-132, 1952.
39. Barsotti, M., and Vigliani, E. C. Bladder lesions due to aromatic amines: statistics and prophylactic measures. *Med. d. lavoro* **40**:129-137, 1949.
40. Truhaut, R. Chemical causes of occupational cancers: aromatic amines acting on bladder. *Arch. Indust. Hyg.* **5**:264-273, 1952.
41. Eisenbud, M. Radiation hazards in atomic energy program. *Indust. Med.* **20**:7-11, 1951.
42. Morgan, K. Z. Historical sketch of radiation protection experience and increasing scope of radiation protection problems. *Indust. Med.* **20**:148-156, 1951.
43. Hempelmann, L. H., Lisco, H., and Hoffman, J. G. Acute radiation syndrome: study of 9 cases and review of problem. *Ann. Int. Med.* **36**:283-510, 1952.
44. Hempelmann, L. H. Examples of acute radiation syndrome in man. *New Eng. J. Med.* **246**:776-782, 1952.
45. Chamberlain, A. C. Turner, F. M., and Williams, E. K. Evaluation of white-cell counting in radiation protection. *Brit. J. Radiol.* **25**: 169–176, 1952.
46. Knowlton, N. P., Jr. Changes in blood of humans chronically exposed to low level gamma radiation. LADC #587, U. S. Atomic Energy Com. 1948.
47. Dickie, A., and Hempelmann, L. H. Morphologic changes in lymphocytes of persons exposed to ionizing radiation. *J. Lab. & Clin. Med.* **32**:1045-1059, 1947.
48. Ingram, M., and Barnes, S. W. Confirmation of unusual finding in blood of cyclotron workers. *Science* **113**:32-34, 1951.
49. *Industrial Medicine on the Plutonium Project: Survey and collected papers.* Edited by R. S. Stone. 511 pp. New York: McGraw-Hill Book Company, Inc., 1951.
50. Atomic Energy Commission, United States. *The Effects of Atomic Weapons.* Washington: The Combat Forces Press, 1950.
51. *Atomic Medicine,* Edited by C. F. Behrens. 416 pp. New York: Thomas Nelson & Sons, 1949.
52. Hamilton, J. G. Metabolism of fission products and heaviest elements. *Radiology* **49**:325-343, 1947.
53. Langham, W. Determination of plutonium in human urine. *Biochem. Sect. of Health Div.,* Los Alamos Scientific Lab., University of California, 1948.
54. Lisco, H., Finkel, M. P., and Brues, A. M. Carcinogenic properties of radioactive fission products and of plutonium. *Radiology* **49**: 361-363, 1947.
55. Hodge, H. C. Pharmacologic tools in study of mechanism of uranium poisoning. *Arch. Indust. Hyg.* **2**:300-311, 1950.
56. Hodge, H. C., et al. Antidotal action of polyphosphates in uranium poisoning. *J. Pharmacol. & Exper. Therap.* **101**:17, 1951.
57. White, M. R., and Schubert, J. Action of salts of zirconium and other metals on plutonium and yttrium distribution and excretion. *J. Pharmacol. & Exper. Therap.* **104**:317-324, 1952.
58. Foreman, H. Use of chelating agents for accelerating excretion of radioelements. *UCRL 1604,* Feb. 28, 1952.

59. Martland, H. S., Conlon, P., and Knef, J. P. Unrecognized dangers in use and handling of radioactive substances. *J. A. M. A.* **85**: 1769-1776, 1925.

60. Evans, R. D. Protection of radium dial workers and radiologists from injury by radium. *J. Indust. Hyg. & Toxicol.* **25**:253-269, 1943.

61. Aub, J. C., Evans, R. D., Hempelmann, L. H., and Martland, H. S. Unpublished data.

62. Progress report of clinical studies on twenty-four patients by radium toxicity group, Div. of Biolog. & Med Res. Argonne National Lab. Edited by W. B. Looney. *ANL 4066*, July, 1951.

63. Hunter, F. T., Merrill, O. E., Trump, J. G., and Robbins, L. L. Protection of personnel engaged in roentgenology and radiology. *New Eng. J. Med.* **241**:79-89. 1949.

64. Daland, E. M. Radiation damage to normal tissues in diagnosis and treatment of nonmalignant conditions and its surgical repair. *New Eng. J. Med.* **244**:959-964, 1951.

65. Peller, S., and Pick, P. Leukemia and other malignant diseases in physicians. *J. A. M. A.* **147**:893, 1951.

66. Muth, H., and Roth, H. H. Results of physical measurements in case of occupational radium poisoning with fatal outcome. *Strahlentherapie* **80**:271-280, 1949. (*Brit. J. Indust. Med.* **8**:108, 1951. Abstr.)

67. Berman, I. L., and Ernest, E. P. Use of radioactive static eliminators in printing plant. *Indust. Med.* **19**:229, 1950.

68. Bourne, H. G., and Cordier, E. J. Public health aspects of industrial x-ray apparatus in Ohio. *Indust. Med.* **21**:21-24, 1952.

69. Cogan, D. G. Lesions of eye from radiant energy. *J. A. M. A.* **142**: 145-151, 1950.

70. Cogan, D. G., Martin, S. F., and Kimura, S. J. Atom bomb cataracts. *Science* **110**:654, 1949.

71. Cogan, D. G., and Donaldson, D. D. Experimental radiation cataracts. I. Cataracts in rabbit following single x-ray exposure. *Arch. Ophth.* **45**:503-522, 1951.

72. Wright, B. M. "Freshly fractured surface" theory of silicosis. *Nature* (London) **166**:538-540, 1950.

73. Ray, S. C., King, E. J., and Harrison C. V. Action of variable amounts of quartz on lungs of rats. *Brit. J. Indust. Med.* **8**:62-67, 1951. *Ibid.* Action of small amounts of quartz and larger amounts of coal and graphite on lungs of rats. **8**:68-73, 1951. *Ibid.* Action of anthracite and bituminous coal dusts mixed with quartz on lungs of rats. **8**:74-76, 1951.

74. Bedford, T., and Warner, C. G. Size and nature of dust particles found in lung tissue. *Brit. J. Indust. Med.* **7**:187-194, 1950.

75. Dreesen, W. C. Pneumoconiosis in United States. In *Proceedings of the Ninth International Congress on Industrial Medicine*. London, September, 1948. Bristol: John Wright & Sons, 1949. Pp. 720-723.

76. Vorwald, A. J., et al. BCG vaccination in silicosis. *Am. Rev. Tuberc.* **62**:455-474, 1950.

77. Brown, E. W., and Van Winkle, W., Jr. Present status of aluminum in therapy and prophylaxis of silicosis. *J. A. M. A.* **140**:1024-1029, 1949.

78. Evans, D., and Schweisheimer, W. Prevention of silicosis. *South African Mining Engin. J.* **60**:807, 1950. (*Arch. Indust. Hyg.* **4**: 92, 1951. Abstr.)

79. Mödder, von H., and Schmitt, T. New manifestation of aluminosis. *Deutsche med. Wchnschr.* **76**:85-87, 1951. (*Arch. Indust. Hyg.* **5**: 89, 1952. Abstr.)

80. Ivanova, M. G., and Ostrovskaja, I. S. Effect of aluminum dust on animal organism. *Gigiena No.* 4, 21, 1950. (*Brit. J. Indust. Med.* **8**:112, 1951. Abstr.)

81. King, E. J., et al. Effect of aluminum on silicosis-producing action of inhaled quartz. *Brit. J. Indust. Med.* **7**:27-36, 1950.

82. Kennedy, B. J., et al. Effect of adrenocorticotropic hormone (ACTH) on beryllium granulomatosis and silicosis. *Am. J. Med.* **10**:134-155, 1951.

83. Meyer, F., and Solomon, S. X-ray diffraction study of sputum in silicosis. *Arch. Indust. Hyg.* **4**:443-445, 1951.

84. Meiklejohn, A. History of lung diseases of coal miners in Great Britain: I. 1800-1875. *Brit. J. Indust. Med.* **8**:127-137, 1951.

85. *Idem.* History of lung diseases of coal miners in Great Britain: II. 1875-1920. *Brit. J. Indust. Med.* **9**:93-98, 1952.

86. McCallum, R. I. Pneumoconiosis of coal miners in North East England with special reference to Durham coalfield. *Brit. J. Indust. Med.* **9**:99-107, 1952.

87. Cochrane, A. L., Fletcher, C. M., Gilson, J. C., and Hugh-Jones, P. Role of periodic examination in prevention of coalworkers' pneumoconiosis. *Brit. J. Indust. Med.* **8**:53-61, 1951.

88. Meiklejohn, A. Doctor and workman. *Brit. J. Indust. Med.* **7**: 105-116, 1950.

89. Corcoran, W. J. Roentgenologic aspects of anthracosilicosis. *Pennsylvania M. J.* **53**:505, 1950.

90. Fletcher, C. M., and Oldham, P. D. Use of standard films in radiological diagnosis of coalworkers' pneumoconiosis. *Brit. J. Indust. Med.* **8**:138-149, 1951.

91. Cochrane, A. L., Davies, I., and Fletcher, C. M. "Entente radiologique." Step towards international agreement on classification of radiographs in pneumoconiosis. *Brit. J. Indust. Med.* **8**:244-255, 1951.

92. Garland, L. H., and Cochrane, A. L. International test of chest roentgenogram interpretation. *J. A. M. A.* **149**:631-634, 1952.

93. Gough, J., James, W. R. L., and Wentworth, J. E. Comparison of radiological and pathological changes in coal workers: pneumoconiosis. *J. Fac. Radiologists* 1:28-39, 1949. (*Arch. Indust. Hyg.* **4**:91, 1951. Abstr.)

94. Motley, H. L., et al. Impairment of pulmonary function in anthracosilicosis. *Arch. Indust. Hyg.* **1**:133-159, 1950.

95. Motley, H. L., Lang, L. P., and Gordon, B. Studies on respiratory gas exchange in 100 anthracite coal miners with pulmonary complaints. *Am. Rev. Tuberc.* **61**:201-225, 1950.

96. Motley, H. L. Pulmonary function studies in bituminous coal miners. *West Virginia M. J.* **46**:8-23, 1950.

97. Slesinger, H. A. Anthracosilicosis in bituminous coal miners: clinical and pathologic manifestations. *Arch. Indust. Hyg.* **2**:284-299, 1950.

98. Gough, J. Paper presented before the Fourth Conference of McIntyre Research Foundation on Silicosis, Norland, Quebec, January 1952.

99. Heppleston, A. G. Coal workers' pneumoconiosis: pathological and etiological considerations. *Arch. Indust. Hyg.* **4**:270-288, 1951.

100. Gordon, B., and Motley, H. L. Pathological and physiological factors involved in treatment of silicosis in coal miners. *Arch. Indust. Hyg.* **2**:365-373, 1950.

101. Motley, H. L., and Tomashefski, J. F. Treatment of chronic pulmonary disease with intermittent positive pressure breathing. I. Evaluation by objective physiological measurements. *Arch. Indust. Hyg.* **5**:1-9, 1952.

102. Vorwald, A. J., Durkan, T. M., and Pratt, P. C. Experimental studies of asbestosis. *Arch. Indust. Hyg.* **3**:1-43, 1951.

103. Shaver, C. G. Further observations of lung changes associated with manufacture of alumina abrasives. *Radiology* **50**:760-769, 1948.

104. Hogue, W. L. Jr., and Mallette, F. S. Study of workers exposed to talc and other dusting compounds in rubber industry. *J. Indust. Hyg. & Toxicol.* **31**:359-364, 1949.

105. McLaughlin, A. I. G., Rogers, E., and Dunham, K. C. Talc pneumoconiosis. *Brit. J. Indust. Med.* **6**:184-194, 1949.

106. Jaques, W. E., and Benirschke, K. Pulmonary talcosis with involvement of stomach and heart. *Arch. Indust. Hyg.* **5**:451-463, 1952.

107. McLaughlin, A. I. G. Talc pneumoconiosis. *Arch. belges de méd. sociale et d'hyg. Médecine du Travail et Médecine Legale*, **8**:451-460, 1950.

108. Troisi, F. M. Silicatosis of micronodular type among women employed in glaze spraying on metal objects. *Indust. Med.* **21**:47-49, 1952.

109. Dunner, L., and Bagnall, D. J. T. Pneumoconiosis in graphite workers. *Brit. J. Indust. Med.* **7**:100, 1950. (Abstr.) (*Brit. J. Radiol.* **22**:573, 1949.)

110. Harding, H. E., and Oliver, G. B. Changes in lungs produced by natural graphite. *Brit. J. Indust. Med.* **6**:91-99, 1949.

111. Jaffé, F. A. Graphite pneumoconiosis. *Am. J. Path.* **27**:907-923, 1951.

112. Parmeggiani, L. Graphite pneumoconiosis. *Brit. J. Indust. Med.* **7**: 42-45, 1950.

113. McCord, C. P. Graphite (plumbago, black lead) as source of dusty lung disease. *Indust. Med. & Surg.* **18**:483-486, 1949.

114. Vorwald, A. J., et al. Diatomaceous earth pneumoconiosis. In *Proceedings of the Ninth International Congress on Industrial Medicine*, London, September, 1948. Bristol: John Wright & Sons, 1949. Pp. 726-741.

115. Vigliani, E. C. Diatomaceous earth silicosis. In *Proceedings of the Ninth International Congress on Industrial Medicine*, London. September, 1948. Bristol: John Wright & Sons,1949. Pp. 742 and 743.

116. Harding, H. E., and Massie, A. P. Pneumoconiosis in boiler scalers. *Brit. J. Indust. Med.* **8**:256-264, 1951.

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

BENJAMIN CASTLEMAN, M.D., *Editor*

VIRGINIA W. TOWNE, *Assistant Editor*

CASE 38391

PRESENTATION OF CASE*

An eighty-year-old widow entered the hospital because of weakness and a heavy feeling in the chest.

The patient, who had awakened in the morning at the usual time, felt faint while preparing breakfast and experienced a depressed sensation over the precordium. After drinking a cup of coffee she returned upstairs to her room. A sister, who lived with the patient, found her several minutes later lying on the floor conscious and complaining of no pain. She had vomited a small amount of bile-stained material. The patient was able to get up and go to bed unassisted. When she was seen by her physician a short time later, the pulse rate was 40, and the blood pressure was 60 systolic. She continued to complain of weakness and a heavy feeling in her chest; she did not complain of headache or dizziness. She entered the hospital three hours after the episode.

The patient had been well and active, and worked part time as a secretary. When she was first seen at this hospital thirteen years prior to admission because of a cold, the blood pressure was 160 systolic, 120 diastolic. She had a sling operation for hallux valgus one year before the present admission, when the blood pressure was 200 systolic, 150 diastolic.

Physical examination showed an oriented but slightly drowsy woman lying flat in bed. The neck veins were slightly distended when the patient was supine but collapsed when she was raised. The fundi showed slight arteriovenous nicking. The lungs were hyperresonant, with fine crackling rales at both bases. The heart sounds were poor. A roughened first sound was heard over the precordium, especially at the third left interspace. There was no peripheral edema. The popliteal pulses were palpable, but the dorsalis-pedis pulses could not be felt.

The temperature was 98.4°F., the pulse 60, and the respirations 22. The blood pressure was 70 systolic, 40 diastolic.

The urinary sediment contained a few white cells per low-power field, but the specimen was otherwise normal. Examination of the blood revealed a hemoglobin of 12 gm. and a white-cell count of 15,000, with 90 per cent neutrophils. An electrocardiogram showed a rate of 75, a PR interval of 0.12 second and a QRS interval of 0.06 second, with normal voltage and axis. There were depressed ST segments and diphasic T waves in Leads 1 and AVL, inverted T waves in Leads 2, 3, V_4, V_5 and V_6 and high R waves in Leads 4, 5 and 6.

The patient vomited several times after admission, raising bile-stained material. The pulse and blood pressure seemed stabilized, but the patient died while being examined four hours after entry.

DIFFERENTIAL DIAGNOSIS

DR. ALFRED KRANES†: I suppose the last sentence that the pulse and blood pressure seemed stabilized means stabilized at this very low level.

DR. ROBERT H. FENNELL, JR.: That is correct; it remained at that low level.

Dr. KRANES: I do not know what this patient had, but I think one comment is indicated. This case illustrates the benign nature of essential hypertension in many patients. The patient was known to have had a diastolic pressure of 120 for at least thirteen years, and probably longer. Women, in particular, may go on for many many years with hypertension and need, therefore, not be subjected to a great deal of therapy. That she eventually died at the age of eighty should not be disturbing.

Patients who die seven hours after the onset of their symptoms are likely to be a diagnostic problem. Certainly, that is the case here. All I know about this woman is that she had slight discomfort in the chest, went into shock and died seven hours later, with an essentially normal or nondiagnostic electrocardiogram and a white-cell count of 15,000. With such meager information, I do not see how it is possible to do more than guess about what may have been going on.

Patients of this age who die within so short a period are most likely to have a vascular accident of some sort, either an occlusion or a rupture of a vessel somewhere. The vascular accident must have been in the chest, the head or the abdomen. There was no clinical evidence of any vascular disturbance in the head; furthermore, it is unusual for a patient with any type of cerebrovascular accident to go into shock. There was nothing to point to the abdomen except the vomiting. Since vomiting is a nonspecific symptom, I do not see how I can put any reliance on that as a localizing sign. I am left with the slight discomfort in the chest, described as a depressed sensation at one time and as a heavi-

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ness at another. This discussion therefore resolves into the types of intrathoracic vascular accidents. Ninety-five times out of a hundred a patient who has slight chest discomfort, goes into shock and dies within a few hours has a coronary occlusion. That may very well be what this patient had. But there are two other conditions that also deserve discussion: one is massive pulmonary embolism, and the other is rupture of an aortic dissecting aneurysm. So far as I can tell from the information supplied, those are the only conditions I need to consider seriously.

The first possibility, massive pulmonary embolism, is supported by little evidence — that is also true, for that matter, of the other diagnoses I am considering. The only evidence to support massive pulmonary embolism is the discomfort and the fact that the patient went into shock; the objections to the diagnosis — and none of this rules it out, since many people with pulmonary embolism do not have any of the signs or symptoms — I shall list. There was no evidence of involvement of the leg veins, which are the most common source of emboli; there was no striking distention of neck veins, which one would expect in a patient with as massive a pulmonary embolism as this would have been. Furthermore, the slow pulse bothers me a good deal. It is conceivable that the apical pulse was not actually 40 when this woman was first seen; if the pulse rate was 40, that would be against a pulmonary embolism.

Dr. F. Dennette Adams: The electrocardiogram reported a rate of 75.

Dr. Kranes: The electrocardiographic rate was 75, but she may have had transitorily a pulse rate of 40; we have no way of determining that.

Dr. Fennell: Several examiners found that she did have a slow pulse on admission. The information concerning her condition at the onset of her illness came directly from the private physician.

Dr. Kranes: Finally, there was no electrocardiographic evidence of pulmonary embolism; such evidence is found only in the more massive infarctions. which is what I should expect in this case. There were no changes characteristic of acute cor pulmonale. The electrocardiogram is essentially negative; its pattern is that of left-ventricle strain and nothing more. For all these reasons I shall have to discard the probability of a massive pulmonary embolism.

Most people with this history would have, if not myocardial infarction, at least a coronary occlusion; infarction may or may not be present in so short a time. Certainly, nothing in the history or in the findings could rule it out. One would expect to find more pain than was described in this patient, but all of us have seen patients with large myocardial infarcts and very little pain. I might add that the meagerness of the pain would be an objection to whatever diagnosis I might consider. The electrocardiogram was certainly not helpful; it did not support the diagnosis of myocardial infarct or coronary occlusion. It was taken only a few hours after the onset of symptoms; perhaps if it had been repeated the next day, more characteristic changes would have appeared. But I can use only the evidence I am given, and I am not presented with any evidence of myocardial infarction. The clinical picture is certainly in keeping with such a diagnosis.

The third possibility is dissecting aneurysm of the aorta. I do not know how to make the diagnosis on the information presented. It is not common for a case of dissecting aneurysm to present with as little discomfort or pain as was described here. Usually, the pain is much more striking, rather prolonged and continuous, and not only is substernal but characteristically radiates to the back, to the abdomen and even to the neck. This patient had none of that. On the other hand, we have seen a number of patients with aortic dissections who have very little pain. The prolonged elevation of the blood pressure would be an adequate background for medionecrosis cystica of the aorta, which these patients usually have. If she did have a dissecting aneurysm, the terminal episode was most probably due to rupture into the left pleural cavity or pericardium. If we had been privileged to observe this patient over a longer period and found that the electrocardiogram remained normal, we would think more seriously about an aortic dissection. When I am presented with an electrocardiogram taken only a few hours after the onset of the illness I cannot place too much diagnostic stress on it. I am, therefore, left more or less where I began. I think that pulmonary embolism would be much less likely than either of the other two, and will leave it as a tossup between myocardial infarction and dissecting aneurysm.

If I were taking care of this patient, there is little doubt that my diagnosis would have been coronary occlusion, and I suspect that is what the clinicians thought in this case; but when these problems are discussed in a clinicopathological conference one's reasoning is somewhat different than when one is actually facing the patient. Here a case is much less likely to be a clear-cut problem; there is usually a peculiar twist to it. For that reason, a dissecting aneurysm must be seriously considered.

Dr. Adams: Dissecting aneurysm seems slightly less likely on the basis of the character of the pain. Do you think the fact that she had two episodes that evening, went into shock and then died while having an examination is against aneurysm? With a dissecting aneurysm should she have died after the first episode or else recovered for some length of time?

Dr. Kranes: No; I do not think so. Many of these patients are thrown into shock by the initial dissection and yet live a matter of days, weeks or

even months; the terminal episode is the rupture through the adventitia into the pericardial or left pleural cavity.

A PHYSICIAN: Do you think the pulse rate supports the diagnosis of a myocardial infarction?

DR. KRANES: I am glad you brought that out. I meant to say that the fact that the patient had a pulse rate of 40 would be somewhat in favor of an intracardiac lesion.

CLINICAL DIAGNOSES

Coronary occlusion, with myocardial infarction.
?Cerebrovascular accident.
Hypertensive heart disease.

DR. KRANES' DIAGNOSIS

?Dissecting aneurysm of aorta.
?Coronary occlusion, with myocardial infarction.

ANATOMICAL DIAGNOSES

Dissecting aneurysm of aorta, with rupture into pericardium.
Hemopericardium.
Cardiac hypertrophy, hypertensive type.

PATHOLOGICAL DISCUSSION

DR. FENNELL: We were limited at the autopsy to the opening and examination of the chest. Fortunately, that gave us the answer. There was a dissecting aneurysm. The dissection started at a point about midway in the arch, with a 3-cm.-long, longitudinal tear, and was primarily retrograde. The dissection went along the coronary arteries but did not occlude them. It ruptured into the pericardial cavity, causing a massive hemopericardium. Microscopically, the aorta showed typical cystic medionecrosis. This very commonly shows simply the accumulation of basophilic material, or there may be cyst-like spaces.

The heart was enlarged; it weighed 480 gm., which is very large for a patient this size. The left ventricle was thickened, measuring almost 2 cm. There was no evidence of infarction of the heart.

DR. KRANES: How far down the thoracic aorta did it dissect?

DR. FENNELL: Only several centimeters beyond the tear; it did not quite reach the level of the subclavian artery.

A PHYSICIAN: What about the carotid arteries?

DR. FENNELL: They were not involved. The dissection extended up near them but did not occlude them.

DR. KRANES: It is interesting that a process of this sort could occur with so little discomfort. The overwhelming majority of such patients have agonizing pain. The last patient I saw with a very extensive dissection had only a little indigestion. This woman was in shock, and may have been apathetic enough so that the pain was obscured.

DR. FENNELL: She was quite lucid and gave the history in detail.

DR. KRANES: What was the clinical diagnosis?

DR. FENNELL: Coronary occlusion, with myocardial infarction.

CASE 38392

PRESENTATION OF CASE*

A seventy-two-year-old man was admitted to the hospital because of swelling of the abdomen and legs.

For a few years he had been exhibiting signs of senility: euphoria, loss of memory and periods of confusion. He was less active for the two months prior to admission. Three weeks before entry he complained of aching in his back. Five days before admission he noted that his belt was getting very tight. His physician told him he was "filling up with water" and gave him ammonium chloride, without any noticeable effect. He had not noted light stools or dark urine, and there had been no abdominal pain, nausea or vomiting. He had been in the habit of having 1 to 4 cocktails daily for many years. His appetite had been poor for a few years and almost nonexistent for the six months before entry.

Seven months before entry he was in the hospital for excision of a varicose ulcer of his leg; examination at that time showed an abnormal electrocardiogram with arrhythmia due to a shifting pacemaker, auricular and ventricular premature beats and a left-bundle-branch type of intraventricular block; the nonprotein nitrogen was 26 mg., the total protein 6.0 gm. and the hemoglobin 12.2 gm. per 100 cc. One year before admission a hypermature cataract was removed from the left eye.

Physical examination revealed an elderly, deaf, moderately confused but cheerful man. There was an iridectomy scar in the left eye. The neck veins were flat. The chest was widened in the anteroposterior diameter and flared on the right. There was dullness to percussion above the right costovertebral angle. The breath sounds were diminished over the entire thorax, but there were no rales. There was a Grade II apical systolic murmur. The abdomen was protuberant, tense and distended. The liver edge was felt eight fingerbreadths below the costal margin in the right midclavicular line; it was slightly tender and firm. There was ++++ pitting edema of the legs to the groin. There were brown indurated, scaling areas over both anterior tibial crests.

The temperature was 99°F., the pulse 92, and the respirations 24. The blood pressure was 110 systolic, 80 diastolic.

The urine had a specific gravity of 1.013 and gave a + reaction for albumin. Examination of the blood

*Presented at a graduate course in internal medicine.

revealed a white-cell count of 7600, with 84 per cent neutrophils, and a hemoglobin of 10.7 gm. The nonprotein nitrogen was 30 mg., the total protein 5.89 gm., the albumin 3.80 gm., the globulin 3.69 gm., the bilirubin 1.6 mg. direct and 2.8 mg. total, and the serum cholesterol 134 mg. per 100 cc. The prothrombin time was 21 seconds (normal, 15 seconds). A bromsulfalein test showed 42 per cent retention of the dye. A blood Hinton test was negative. A stool guaiac test was negative. Roentgenograms of the chest showed marked elevation of both leaves of the diaphragm and fluid in the right costophrenic sinus, with blunting of the costophrenic angle. There were plate-like areas of atelectasis in both lower lobes. The heart demonstrated some prominence of the left ventricle, and the aorta was tortuous. An electrocardiogram showed no significant change from the previous record.

The patient gradually became increasingly drowsy. His appetite was poor, and it was difficult to maintain an adequate caloric intake. The abdomen became more tense and distended, and the neck veins became distended but did not pulsate. There was a poor response to Mercuhydrin, and the urinary output dropped to 450 cc. per day. On the thirteenth hospital day an abdominal paracentesis yielded 1700 cc. of turbid, cream-colored, slightly orange fluid with a specific gravity of 1.009; the fluid contained 14,460 red cells and 337 white cells per cubic millimeter, of which 188 were neutrophils, and 1.7 gm. per 100 cc. of protein. No malignant cells were seen on cytologic examination of the fluid and there was no growth on culture. After the paracentesis the patient was somewhat improved and was able to sit up for a few days. Then the abdominal fluid quickly reaccumulated, and he lost weight and became weaker. The blood nonprotein nitrogen rose to 94 mg., and the total protein to 6.88 mg. per 100 cc., with an albumin-globulin ratio of 0.6.

By the thirtieth day he was ashen gray and cyanotic, requiring frequent tracheal suctioning to maintain a clear airway; examination of the chest showed loud rhonchi and rales. He became unresponsive and died later on this day.

Differential Diagnosis

Dr. Victor G. Balboni*: May I see the x-ray films?

Dr. J. L. Sosman: The only film we have is a single film of the chest taken in the upright position early in the hospital course, probably prior to the abdominal tap. I do not think I can add anything to the findings described. Both leaves of the diaphragm are quite high; there is plate-like atelectasis above each leaf. The heart is in transverse position; I think there is enlargement of the left ventricle as well. I should consider this a hypertensive type of heart although the blood pressure was not elevated on admission. The aorta is quite tortuous and elongated. There is a small amount of fluid in each costophrenic angle. Nothing is seen in the lungs that is not explained by the presence of ascites elevating the diaphragm. The patient has too much ascites for me to determine whether the spleen is enlarged or not. I cannot make out the splenic border.

Dr. Balboni: In summary, a seventy-two-year-old man came into the hospital with ascites and pleural effusion. On admission the neck veins were not distended, the heart rate was within normal limits, and there were no signs of congestive heart failure. Although there was fluid in both pleural spaces, no rales were heard; therefore, I think I can eliminate cardiac failure as the cause of the ascites and pleural effusion. He was found to have a huge liver; no mention was made of the presence or absence of an enlarged spleen.

Dr. David Kahn: The spleen was not palpable.

Dr. Balboni: That may have been because he had so much abdominal fluid. It would be of great interest to know whether the spleen was or was not enlarged. The liver-function tests on entry showed that he had a chemical jaundice, the total bilirubin being 2.8 mg. per 100 cc. The prothrombin time was reduced, as one would expect in liver disease. The bromsulfalein test was very poor, 42 per cent in the serum at the end of the test period. The serum albumin was somewhat low so that the albumin-globulin ratio was less than 1. It seems fair to assume that this man had severe liver disease on the basis of the tests and the physical findings. The clinical course was that of progressive liver failure, with development of drowsiness and finally coma and death.

The real problem is the cause of the liver failure. From the history I know that the patient had partaken fairly liberally of alcohol, and his appetite had been extremely poor for a few years and nonexistent for six months — an ideal setup for the development of portal cirrhosis. The most widely accepted opinion is that the alcohol is not itself toxic but that cirrhosis is likely to develop in people who drink continuously and who do not have a good, balanced diet. The deficiency of protein in the diet of alcoholic patients, especially the aminoacids methionine and cystine, and also the deficiency of the vitamin B complex are more important factors in the development of cirrhosis than the alcohol per se.

A few things, however, do not fit in too well with a diagnosis of uncomplicated cirrhosis. In the first place, the abdominal fluid is described as turbid, cream colored, slightly orange, with a specific gravity of 1.009, and containing many red cells and some white cells. From that description of the fluid, I should have to call this a chylous ascites or a pseudochylous ascites. A chylous ascites is extremely unusual in uncomplicated portal cirrhosis; a pseudochylous ascites can occur. A pseudochylous ascites develops in patients usually with long-standing

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ascites in which there is a fatty degeneration of cells, giving an appearance of a chylous fluid. Such a fluid, although it contains fat, does not contain the amount of fat found in a true chylous ascites. I should like to ask Dr. Kahn if any specific tests for fat were done on the ascitic fluid.

DR. KAHN: No; apparently, the fluid did not arouse suspicion in the physicians handling the patient.

DR. BALBONI: If I can believe the statement in the history that this man complained of only five days of abdominal swelling, the short duration of the ascites is against pseudochylous fluid; therefore, I am forced to say there was a true chylous effusion. The number of red cells in the fluid is much against cirrhosis — unless the tap was traumatic. For these reasons, I think this man did not have plain alcoholic cirrhosis, and I shall proceed to the possibility that some lesion obstructed the intestinal lymphatic system or the thoracic duct, thus releasing chyle into the peritoneal cavity.

Cancer in the region of the pancreas could give this picture, and then could metastasize to the liver, causing the large liver, with failure. Another possibility that I cannot eliminate is hepatoma, a carcinoma superimposed on a cirrhotic liver. We know that hepatomas, by and large, develop in the presence of cirrhosis of the liver. This man had the setup for cirrhosis and possibly had a cirrhosis, with the subsequent development of a hepatoma.

I should like to make a diagnosis of death due to liver failure, with chylous ascites, both the chylous ascites and the liver disease being due to a malignant lesion, probably in the pancreas, possibly in the liver. In these cases there is always the possibility of an underlying cirrhosis — a possibility I cannot rule out.

DR. KAHN: Did the liver-function tests seem inconsistent with metastatic disease of the liver alone?

DR. BALBONI: I do not think so. The liver-function tests mentioned were fair except for the excretory function, which was poor.

DR. F. DENNETTE ADAMS: That feature makes one hesitate to make a diagnosis of a malignant tumor alone. My conception is that, except in the presence of jaundice, almost the entire liver must be replaced by malignant tissue before the liver-function tests are seriously impaired. If this man had had more jaundice, along with the enlargement and the impaired liver function, that would have been the picture of a malignant process. His liver was much larger than one usually encounters in the terminal stage of cirrhosis. Furthermore, once he began to fail, he went downhill more rapidly than the usual patient with cirrhosis. I think the weight of evidence is in favor of a malignant lesion, probably primary in the pancreas and, if not there, in the liver itself. In such obscure cases lymphoma is always a possible diagnosis, but there was no evidence of lymphoma in this particular patient. Cirrhosis with superimposed hepatoma is also a decided possibility. The fact that in the short time he was in the hospital the patient lost weight so rapidly is characteristic of pancreatic disease.

A PHYSICIAN: He also had an aching in the back.

DR. BALBONI: That is a good point; pain in the back would go with carcinoma of the pancreas.

A PHYSICIAN: Were any serum amylase determinations done?

DR. KAHN: No.

DR. BALBONI: My impression is that the only time a serum amylase value helps is in the early stage of an acute pancreatitis.

A PHYSICIAN: I should add cirrhosis of the liver to cancer, because of the long story of alcoholism, the poor food intake and the avitaminosis.

A PHYSICIAN: How frequently is blood found in the ascitic fluid with hepatoma?

DR. BALBONI: Blood in the ascitic fluid is strong evidence against a simple cirrhosis of the liver, but one would have to be fairly sure the tap was not bloody. Blood in the fluid in the abdomen is due to a malignant process until proved otherwise. The exact percentage of hepatomas that cause bloody ascites is extremely high.

DR. KAHN: The blood would have been found in the ascitic fluid only after the tumor involved the capsule of the liver or if there were peritoneal implants.

A PHYSICIAN: Was the specific gravity of the ascitic fluid more in line with pseudochylous ascites?

DR. BALBONI: Yes; it should be higher in a true chylous ascites.

A PHYSICIAN: Was not the white-cell count of 7600 somewhat against cirrhosis?

DR. BALBONI: Yes, I think so; with cirrhosis there is often splenomegaly, and with splenomegaly there is usually leukopenia.

DR. KAHN: How does one account for the rapid reaccumulation of the fluid?

DR. ADAMS: Rapid reaccumulation usually occurs in cirrhosis, but I believe peritoneal implants or lymph-channel obstruction by tumor could cause it.

DR. BALBONI: The patient had serious liver disease, with portal obstruction as well as obstruction to the lymph nodes.

CLINICAL DIAGNOSES

Laënnec's cirrhosis.

Cerebral arteriosclerosis.

DR. BALBONI'S DIAGNOSES

Liver failure, with chylous ascites.

Carcinoma of pancreas, with metastases to liver.

?Cirrhosis, with superimposed hepatoma.

Anatomical Diagnoses

Mixed hepatoma and cholangioma, with metastases to lung and lymph nodes.
Cirrhosis of liver, alcoholic type.
Ascites.
Portal-vein thrombosis.
Splenomegaly.
Esophageal varices.

Pathological Discussion

Dr. Kahn: At autopsy the peritoneal cavity contained over 12 liters of fluid; this was orange and opalescent, and no further examination of it was carried out. The fluid did not suggest a chylous fluid. The liver weighed 2000 gm. and was diffusely nodular, firm and fibrous; superimposed on this general nodularity were small, white, somewhat softer nodules scattered throughout the entire liver but in heaviest concentration in the left lobe. Microscopically, there was a fairly typical Laënnec's cirrhosis with abundant "alcoholic" hyaline. Superimposed on this was a primary carcinoma of the liver of an unusual type — a mixed hepatoma and cholangioma. This carcinoma had invaded the radicles of the portal and hepatic veins, and was diffusely spread through every section of the liver that I examined. In most of the growth the tumor cells were arranged in cords and rows simulating to some extent liver tissue; in other areas the tumor cells formed glandular spaces lined by tall, columnar cells, some of which were mucous secreting, resembling a cholangioma or primary carcinoma of the bile ducts.

The mixed type of tumor in the liver is said to be somewhat more malignant than either pure hepatoma or pure cholangioma. In all reported cases of tumor of this type autopsy disclosed metastases to the lung, and this was no exception; there was a metastasis to the right middle lobe of the lung, as well as to the lymph nodes in the lesser omentum, around the head of the pancreas and in the periaortic nodes. The association of hepatoma and cirrhosis is well known. In all large series reported 75 per cent of hepatomas and 50 per cent of cholangiomas occurred in association with cirrhosis.

A third finding of interest in this case was thrombosis of the portal vein. This thrombus partially occluded the main portal vein and the terminal parts of the superior mesenteric and splenic veins. There was moderately advanced organization of the thrombus, indicating that it had been present for some time. We also found evidence of portal hypertension: splenomegaly (the spleen weighed 380 gm.) and a moderate number of esophageal varices. The rapidly recurring ascites, then, was probably caused by any one of, or a combination of, the following: blockage of radicles of the portal vein by carcinoma, partial occlusion of the portal vein and blockage of the mesenteric lymphatics by tumor.

A Physician: Have you seen carcinomas after infectious hepatitis?

Dr. Kahn: Although it is the accepted opinion that postnecrotic cirrhosis develops in some cases after infectious hepatitis, there is only an occasional well documented case of such an occurrence. We have seen hepatomas in livers showing postnecrotic cirrhosis, but to my knowledge we have never been able to prove the antecedent infectious hepatitis.

A Physician: Do hepatomas develop in cases of nutritional cirrhosis in the tropics?

Dr. Kahn: Certainly, in some parts of the world, primary carcinoma of the liver is one of the commonest malignant tumors seen, and these are almost invariably found in cirrhotic livers: it is the consensus that the cirrhosis in these countries is on a nutritional basis. Flukes are considered to be the cause of the cirrhosis in some parts of Asia.

The New England Journal of Medicine

Formerly

The Boston Medical and Surgical Journal

Established 1828

Official Organ of
THE MASSACHUSETTS MEDICAL SOCIETY
and
THE NEW HAMPSHIRE MEDICAL SOCIETY
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MATERIAL, other than original articles, should be received not later than noon on Thursday, three weeks before date of publication.

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GREYHOUNDS, LIMITED

EDUCATION, the most important factor in maintaining the world's social and economic equilibrium, needs constant revision. Not only is the sum of available knowledge constantly increasing, but with this increase the technology of its use is becoming more complex. It is with humility that anyone should express his views on education, and yet the more it is discussed, the more likely it is that solutions will be found for many of its problems.

Recent events indicate that a healthy concern over the complexities and the shortcomings of the educational process continues to stir the minds of educators. Momentum is lost in the shifting of the gears, and as with a car plowing through heavy snow, loss of momentum may be fatal to continued progress. Even with education today apparently so much more effective than it was a generation ago, there is still this lost motion. The facts of college life are no longer represented by the stein on the grand piano, but this symbol of higher education has not yet been entirely replaced by the cool intellectual efficiency so admired by the professionals.

The matter of the effective transmittal of the educational impetus from school to college is the chief problem of pedagogy, whose high priests find the greatest loss of power taking place between the eleventh and fourteenth grades. This is a general problem; an immediate concern is that of the "intellectually gifted" youth whose powers, it is believed, are wasted under the present lockstep system.

A study has accordingly been put on foot, sponsored by the preparatory schools at Andover, Exeter and Lawrenceville in conjunction with Harvard, Princeton and Yale colleges, to see what can be done with these academic greyhounds. In line with the study, which the Ford Foundation is underwriting, the Foundation has awarded 200 fellowships to start in college boys sixteen and a half years of age; the College of the University of Chicago is offering to similar superior students the opportunity of completing a basic liberal-arts training two years ahead of the usual time.

The suggestion has been made that through a blending of their now sharply demarcated educational areas, preparatory school and college could between them engineer an accordion-pleated course that would permit the keen in mind to enter college as sophomores. It is agreed that in this experimental study, timed to begin this fall, emotional and social factors will receive consideration. From a medical and social point of view these factors may indeed be considered of even greater importance than the scholastic brilliance on which the selection of the experimental material is likely to be largely based. Experiments in the forced training of youths of ordinary intelligence but superior emotional stability will be fraught with less danger than the application of the methods to boys of high scholastic capacity, but limited in their physical, social, and emotional development.

The starting in college of 200 boys under sixteen and a half years of age is an experiment to be viewed with interest but also with some apprehension, for the period of advanced education is still believed in many quarters to be one in which other forms of development take place than those that come solely or largely through the curriculum. College is still a place where good fellows may get together with mutual benefit, even if their steins leave rings on the grand piano.

Acceleration in education for those who can safely accelerate will have its advantages, if taken in moderation. Enrichment of the academic pabulum offered may be still better for those with good intellectual digestions who are not hasty eaters; a little of the first and much of the second may help young America to preserve the protective coloration that is lost when age patterns are too grossly dislocated.

Two interests in fact caution moderation — that of the training of the 90 and 9 students, less brilliant, who represent nevertheless the rank and file of educated persons, and that of the emotional stability and social well-being of the 1 in 100 on whom the educational experiments are made. For the race is not always to the swift, nor the battle to the strong.

ANTIBIOTIC THERAPY AND FUNGOUS INFECTIONS

THE general impression has become prevalent that patients under treatment with antibiotics, particularly the broad-spectrum antibiotics (aureomycin, terramycin and chloramphenicol) are likely to develop fungous infections. As a result, physicians are loosely referring to a large variety of untoward manifestations incident to antibiotic therapy, including sore tongue, hairy tongue, pruritus ani or vulvae, vaginitis, proctitis and indolent pulmonary infections, as fungous infections — or even more specifically as moniliasis. Physicians who have taken the trouble to study these complications bacteriologically have been supported in this view by the frequent reports of positive smears or cultures for yeast-like organisms, particularly *Candida albicans* from exudates, scrapings or tissues from the affected areas in antibiotic-treated patients. Moreover, numerous reports in the medical literature, some of them citing impressive numbers of cases to which the diagnosis of moniliasis has been attached, and a warning from the Council on Pharmacy and Chemistry of the American Medical Association[1] that such cases are to be expected in patients under prolonged treatment with the broad-spectrum antibiotics have given the weight of authority to this impression.

In a recent paper Kligman[2] bases a critical review of the evidence on this problem not only on a careful evaluation of the reported laboratory and clinical data but also on extensive and thorough original observations in patients, supplemented by studies in vitro and in experimentally infected animals. The paper also contains a thorough discussion of the diagnosis of moniliasis, pointing out specifically the complexities of recognition of pulmonary moniliasis.

Kligman concludes that *C. albicans* regularly emerges in abundance in the mouths and gastrointestinal tracts of persons receiving wide-spectrum antibiotics. The isolation of this organism in the presence of some untoward side-reaction such as glossitis or stomatitis, however, is not tantamount to a diagnosis of moniliasis. His evidence indicates that the stigmatization of the mucous-membrane reactions observed in association with antibiotic therapy as moniliasis is probably unfounded; he also implies that the diagnosis of pulmonary moniliasis has rarely, if ever, been correctly made.

The gist of Kligman's discussion is that although monilia and similar organisms, which are ordinarily saprophytic, are readily identifiable, and sometimes in great abundance in cultures from patients under treatment with antibiotics, their relation to specific symptoms or lesions has only rarely been established. He found no acceptable evidence, either from his own studies or from those reported by others, that the wide-spectrum antibiotics enhance the growth of *C. albicans* in vitro, nor was he able with these antibiotics to potentiate mycotic diseases in animals with experimental moniliasis, blastomycosis, histoplasmosis, coccidioidomycosis and sporotrichosis. From the clinical point of view, the replacement of the respiratory or gastrointestinal flora with antibiotic-resistant strains of pathogenic organisms, such as hemolytic *Staphylococcus aureus* and hemolytic streptococci, is potentially a much more seri-

ous problem than the overgrowth of the normally saprophytic fungi or yeast-like organisms.

References

1. Council on Pharmacy and Chemistry. Warning statement to be included in aureomycin hydrochloride, chloramphenicol and terramycin hydrochloride labelling. *J. A. M. A.* **145**:1267, 1951.
2. Kligman, A. M. Are fungus infections increasing as result of antibiotic therapy. *J. A. M. A.* **149**:979-983, 1952.

CINEMATIC SURGERY

THE London *Lancet* in its most quotable department, "In England Now," (May 24, 1952) quotes — from this side of the water — an analysis of the parts played by physicians in American films, published in a contemporary cisoceanic journal. If, as has been said, shipments of tea benefit from a sea voyage, then this particular reference should doubly benefit from its two crossings, if it bears any analogy to tea.

The reference is purely statistical, any resemblance to known characters, real or imaginary — except possibly Mr. Jekyll and Dr. Hyde — being entirely fortuitous. In 1949, according to the *Lancet's* peripatetic correspondent, 211 physicians were portrayed in 411 films; in 1950, 237 such portrayals occurred in 428 films. In 1949 physicians were cast as mild to severe villains 12 times; in 1950 they were the villains of the plots, more or less, in 13 instances. The point is stressed that these characterizations are entirely personal. When portrayed as a group, physicians were always depicted sympathetically — a result no doubt of those finer feelings that linger in every fiber of the film director's personality.

But the whole thesis is clarified when expressed in tabular form, for those who can read tables:

Year	Sympathetic and Prominent	Sympathetic but Less Prominent	Sympathetic as a Group
1949	22	128	3
1950	21	148	4

It is admitted that these classifications are still a little hard for the statistical laymen to comprehend, although one may assume that the worst of the villains are sympathetically buried in the last, or group column.

Interestingly enough, it is considered bad taste for an American doctor to appear in the film as a comic, and in this, of course, professional dignity will lead all physicians to concur. In 1950 doctors were allowed to appear only twice in humorous roles, tentatively. The much-traveled report does not state whether these laugh producers were urologists, according to the common concept, or others.

It seems that a charter has been granted for the admission of another medical college into the already great brotherhood of physician-making institutions of the country. It is to be located at Milwaukie, but its complete organization may be deferred for the present. . . . It seems morally impossible for all our new medical schools to succeed. Individually, we lament this incessant increase, as it tends to weaken the course of instruction.

Boston M. & S. J., *September 29, 1852*

MASSACHUSETTS MEDICAL SOCIETY

DEATH

MACKAY — George F. MacKay, M.D., of Dalton, died on August 28. He was in his eighty-second year.

Dr. MacKay received his degree from New York University Medical College in 1894. He was a member of the staffs of Pittsfield General and Hillcrest hospitals and a fellow of the American Medical Association.

He is survived by his widow and two brothers.

BOSTON MEDICAL LIBRARY

REPORT OF THE LIBRARIAN, Part II*

MEDICOHISTORICAL ACTIVITIES IN 1951

HENRY R. VIETS, M.D.

BOSTON

A number of works of unusual merit in medicohistorical literature were published in 1951. Four, at least, were outstanding. The first and foremost was the publication of Sigerist's first volume of his monumental *History of Medicine* (New York: Oxford University Press), reviewed by the Librarian in *Centaurus* (1:388-391, 1951). The book covers the period of primitive and archaic medicine, including the beginnings in Egypt and Mesopotamia. Of value, too, are the appendixes on histories of medicine, source books and museums.

Another contribution consisted of the two stout volumes of the *Letters of Benjamin Rush* (Princeton: University Press), skillfully edited by L. H. Butter-

*Presented at the annual meeting of the Boston Medical Library, May 19, 1952.

field. Rush, born in 1746, took his apprenticeship in Philadelphia, graduated from Edinburgh in 1768, visited London and Paris, and returned to Philadelphia to begin practice in 1769. He first taught chemistry but soon became involved in the American Revolution, became a delegate to the Continental Congress and signed the Declaration of Independence. Later he served as surgeon general of the troops between the Hudson and Potomac rivers. At this time he wrote his famous "Directions for Preserving the Health of Soldiers." Rush is also remembered for his account of the epidemic of yellow fever in Philadelphia in 1793. In a long series of letters to his wife, written during the summer and autumn of that year, he provides a graphic day-by-day record of the epidemic. Many of these, reprinted in Butterfield's edition, are in the remarkable collection of the late Josiah C. Trent, of Durham, North Carolina. Rush relied for treatment of the yellow fever on calomel and jalap, 10 gr. (0.6 gm.) of each, as a mercurial purge, an idea he had picked up in the army hospitals. This regimen became known as "Rush's ten-and-ten." Later the jalap was increased to 15 gr. (1 gm.) "to carry the calomel through the bowels." Rush, as has often been said, was an observant man but not a good observer. His chief weakness in his report on yellow fever is his failure to keep exact records of his own cases, but it was not easy to do so because of the rapid onset of the epidemic. His fondness for depletion as the chief aim of treatment led him to extremes in practice, but he could report to his wife in September: "I now save 29 out of 30 of all to whom I am called on the first day, and many to whom I am called after it." To prevent the disease he advised keeping the bowels gently open, "for in them the disease first fixes its poison." Rush often saw 100 patients a day. During this harrowing experience he lived on a diet of vegetables and drank nothing but water. The demands on his time became so heavy that he was unable to comply with all the calls and consequently gave advice through the press on both depletion by purge and by bloodletting. Infected himself, he was quickly cured by his own remedy, but he could not save his "dear and amiable pupil" Johnny Stall, aged nineteen, or two other apprentices and his sister. He resigned from the College of Physicians on account of his disagreement over the treatment of yellow fever, but he was made professor of medicine at the University of Pennsylvania in 1796. His later life was given over to the publication of his books and service to the United States, particularly in bringing about a reconciliation between John Adams and Thomas Jefferson, but of most interest medically are a wonderful series of letters to his son James, written to Edinburgh, where the boy was following his father's footsteps in medicine.

The third book of note was *A Bio-bibliography of Edward Jenner, 1749–1823* (London: Harvey and Blythe) by W. R. LeFanu, the distinguished librarian of the Royal College of Surgeons, London. The book received special comment by the Librarian in the *New England Journal of Medicine* (**245**:348, 1951). To LeFanu's list (No. 33) may now be added the Latin translation of *An Inquiry* (Vienna, 1799), acquired by the Library in 1951.

The fourth book, *The Apologie and Treatise of Ambroise Paré Containing the Voyages made into Divers Places with Many of His Writings upon Surgery* (London: Falcon Educational Books), was edited by the practiced hand of Geoffrey Keynes. Surgery was dominated in the last half of the sixteenth century by the great figure of Paré. It was he who raised this branch of medicine from a despised profession to one of dignity and esteem. Born in 1510, Paré was apprenticed to a barber-surgeon in Paris at twenty-two and served as house surgeon in the Hôtel-Dieu for a number of years before going off to war as an army surgeon. His campaigns are depicted in the *Apologie*, the reprinting of which is the main object of Keynes' volume. It contains what amounts to an autobiography of fifty years of Paré's life. Paré developed in the field the principle of reconstructing the course of a projectile in the body by placing the parts in their position at the time of injury. Returning to Paris, he began an intensive study of anatomy and carried on a practice that included obstetrics. In his anatomic treatise, published in 1549, he described podalic version, an original and important contribution to the subject. Since he knew no Latin, he wrote in French, much to the disgust of the Faculty of Physicians, who considered him an ignorant charlatan, but his *Oeuvres*, issued in 1575, could not be suppressed and the book "took its place as the most enlightened work on surgery ever published." Since previous translations of Paré's contributions to surgery have long been out of print, Keynes' meticulous reprint, with an informative introduction, is particularly welcome.

The passing of the voluntary hospital in England into state ownership in 1948 occasioned a number of histories of solid worth. Among them was the sympathetic account of the general hospital near Nottingham, founded in 1781, by Frank H. Jacob (Bristol: John Wright and Sons). Early physicians were John Storer, John Attenburrow, who began vaccination in Nottingham in 1800, Marshall Hall, who briefly served the hospital, and Alexander Manson, pioneer in the use of iodine for goiter or "Derbyshire neck," in 1821. Later physicians were Francis Sibson, who advanced knowledge of diseases of the chest while a resident surgeon and promptly studied the action of ether and chloroform in 1846, and John Calthrop Williams, who wrote on nervous palpitation of the heart, distinguishing it from the result of organic disease (1836). The most interesting character in the book is not a doctor but the chemist, Jesse Boot, later Sir Jesse Boot and finally

Baron Trent, who was the great benefactor of both the hospital and the University College in Nottingham. A victim of severe arthritis in his middle thirties, he lived to the age of eighty-one, part of the time with legs and arms doubled up and fixed, unable to move from neck to feet, but being carried all over England inspecting his business affairs and his vast philanthropies.

Pleasing, too, is *Guy's Hospital, 1725–1948* (London: Guy's Hospital Gazette Committee), a well illustrated account containing the "History," written in 1925 by Sir William Hale-White and now brought up to date, with additional chapters on the medical and dental schools, nursing and "Guy's Hospital in 1948." The fine east wing of the hospital was destroyed by bombing in 1941. The illustrations, many in color, add greatly to the attractiveness of this brief history.

From the Royal College of Surgeons of England comes *A Record of the Years from 1901 to 1950* (London: Royal College of Surgeons), to honor its one hundred and fifty years of corporate life, following on William MacCormac's two memorial volumes issued in 1900. It presents the lives of all the presidents for the last fifty years, — Godlee, Cheyne, Makins, Bowlby, Bland-Sutton, Lord Moynihan, Webb-Johnson, Wakeley and others, — along with an account of the tragic bombing of 1941 and the reorganizations of recent years, including the gifts of the American College of Surgeons in 1947.

Another book of interest is Peter Lovegrove's *Not Least in the Crusade. A Short History of the Royal Army Medical Corps* (Aldershot: Gale and Polden). Long unorganized, but effective from the seventeenth century, the Corps did not receive a Royal Warrant by Victoria until 1898. Its baptism of fire occurred in the Sudan the same year, and since then the Corps has carried on the highest standards under such well known figures as Keogh, Sloggett, Leishman and Cantlie.

Among the anthologies published in 1951, *Doctors by Themselves* (London: Cassell and Company), by Edward F. Griffith, composed of 869 selections, is one of the best. The author, a London physician, writes that he originally had 5000 excerpts, which he pruned down to make a single volume. The paragraphs culled from physicians' writings are chosen with care. Each is numbered and followed by the name of the contributor and his inclusive dates. From the key number one turns to the index of sources for the exact reference, including the page where the selection occurs. In addition, biographic notes, listed alphabetically, are separately indexed, with the author's numerical contribution. The book is thus usable, comprehensive and inclusive. Portraits of many authors are used as illustrations. Only a few errors seem to have crept into this splendid and timely contribution. Sections deal with such subjects as how the doctor works, amuses himself and looks at life, death and immortality. The doctor's "troubles" are not forgotten.

In a slightly different category is *The Physician as Man of Letters, Science and Action* (Edinburgh and London: E. and S. Livingstone), second edition, by Thomas Kirkpatrick Monro. The original work was first published in 1933, having evolved from an address by the author as president of the Royal Medico-Chirurgical Society of Glasgow in 1932. Concerned with data on medical men who distinguished themselves in other ways than the practice of medicine, Monro lists the medical poets, dramatists, philosophers, saints, philanthropists, lawyers, politicians, ambassadors, governors, soldiers and explorers, as well as those who won a name on the stage, in aeronautics, collecting, art, music, piracy, crime and other forms of endeavor. One chapter deals with American physicians, but Harvey Cushing and others are missing. The biographic notices are uneven, and occasionally in error, but the book in general is useful and covers a wide territory. Of special value is the section dealing with men who were for a time medical students and yet never obtained a degree.

Selected Writings of Sir William Osler (London: Oxford University Press), with an introduction by Geoffrey Keynes, again provides the best of Osler in an attractive form. His memory cannot be better served than by a reading, or a re-reading, of his addresses and essays. Included, of course, are "Books and Men," the biographies of Browne, Patin, Servetus, Burton, Beaumont and Laënnec; "The Student Life"; "The Growth of Truth"; and a chapter, not previously printed in any former collection, "Letters to my House Physicians." The book is edited as a centenary tribute by Alfred White Franklin and a committee of the Osler Club of London. The "Letters," originally printed in the *New York Medical Journal*, are descriptive of medical life in Freiburg, Bern and Munich as seen by Osler in May, 1890. He was traveling with Ramsay Wright, of Toronto, brushing up no doubt for the *Textbook*, to be written and published the next year, and in addition to visiting clinics, the young men went to the Passion Play at Oberammergau. Osler posed the question of why Christ should have died after so comparatively short a time upon the cross. He found a satisfactory solution in the mental and physical exhaustion consequent upon the trials of the preceding twenty-four hours and rejects the theory that death was due to rupture of the heart as advocated by William Stroud in *A Treatise on the Physical Cause of the Death of Christ, and Its Relation to the Principles and Practice of Christianity* (London: Hamilton and Adams, 1847) — a form of collapse "highly improbable in a vigorous, healthy man of thirty-three." Osler, historically minded at forty, as well as clinico-pathologically indoctrinated, also found a long ac-

count of the play written in 1830 by Oken, the anatomist. The presentation in 1890 was unchanged.

Chiefly of value for the biographies of Sir William Knighton, physician to George the Fourth, Sir James Clark, the meteorologist, who cared for John Keats in Rome during the young poet's last illness and later become physician to Queen Victoria, and of Sir William Jenner, the naturalist, also physician to Victoria in a later period, *The Healing Touch* (Springfield, Illinois: Charles C Thomas), by Harley Williams, was first issued in England in 1949. Other chapters are concerned with Florence Nightingale, Brown-Séquard, the Mayo Brothers, Halsted, Cushing and some others, but the best section of the book is on Chadwick and Southwood Smith, the great pioneers of English health reform in the mid-nineteenth century.

Of the biographies of 1951, one of the most enlightening is *The Versatile Victorian. Being the Life of Sir Henry Thompson, Bt. 1820-1904* (London: Harvey and Blythe), by Zachary Cope. Thompson, whose portrait by Millais in the National Gallery and cartoon by "Ape," made his face and figure familiar to many, was a surgeon, artist, novelist, social reformer and traveler, whose "octaves," or dinners for eight persons in the seventies, elevated the giving of dinner parties to a fine art. If one thinks of dining with James Paget, Spencer Wells, Lauder Brunton, William Broadbent, Victor Horsley, Richard Quain and Ernest Hart, one will sense the flavor of what it meant to be invited to an "octave" at Thompson's house in 1872. At the three-hundredth "octave" in 1904, the year of Thompson's death, the Prince of Wales, Joseph Choate, Alma-Tadema, Conan Doyle and others of equal note were present. Thompson wrote on such varied subjects as *Nankin Porcelain*, of which he had an outstanding collection, *Food and Feeding*, a popular book in its day, *Modern Cremation* and *The Motor Car* (he was an early advocate of the automobile in 1902). His professional books were on urology, his special field of practice. The description of a medical student's life in *Charley Kingston's Aunt*, a novel, is clearly autobiographic, for Thompson was a poor boy who had no prospects after the cost of his education had been defrayed. An account of what he did with his life, how famous and honored he became and how generous and sincere a man resulted can be found in the pages of Cope's delightful book, a tribute from one great surgeon to another.

Closer at hand is the famous series entitled *Sibley's Harvard Graduates*, which Clifford K. Shipton continues to edit. The current volume gives biographic sketches of those who attended Harvard College in the classes 1726–1730. As usual, a good company of the graduates practiced medicine. One was William Clark (class of 1726), who studied medicine in London in 1731 but returned to Boston without a medical degree. His preparation being far above the ordinary colonial standards, his practice included the cream of New England society. Besides, he attended the soldiers at Castle William for many years, sold drugs, did not despise pulling teeth and was active in the Boston Medical Society in the mid-eighteenth century. When he died in 1760 he was mourned as "a Physician of great note."

Another was John Prescott (class of 1727), son of a doctor of Concord, who studied medicine with his father. He developed a large practice when only seven years out of college. In 1740 he raised a company of volunteers for royal service in the West Indies. He was with Admiral Vernon on the expedition against Carthagena as a surgeon, as was Tobias Smollett. Returning to Jamaica, the Massachusetts company was almost decimated by yellow fever. In 1743 he went to London to negotiate the war expenses. There, having escaped the deadly "Yellow Jack" in Jamaica, he died of smallpox in the same year. His widow enjoyed a royal pension until her death over fifty years later.

Shipton also relates the story of one of the first American martyrs to science, John Fitch, who graduated from Harvard College in 1728. He was apprenticed to Nathaniel Sargent, of Hampton, also a physician, who had graduated in 1707. In Newbury, where he settled, an epidemic of throat distemper broke out in 1735. For a year Fitch battled the disease, only to die of it in 1736. The *Boston News-Letter* and the *Boston Gazette* both wrote of his being highly regarded as a physician, and Ernest Caulfield has recently paid tribute to him.

Outstanding in the graduates of this period was Ezekiel Hersey of the class of 1728. He studied medicine under Dalhonde, a French physician of Boston and then with Hugh Kennedy, both well known physicians. With the latter he signed an apprenticeship agreement "to Live with . . . Kennedy the Space of two years and to give Said Kennedy the Sum of one hundred pounds for his instructing him in the Knowledge of Physick and Surgery and to find him in meat, drink, washing and Lodging." Kennedy inoculated his apprentice for smallpox, who took the disease very hard, much to the Doctor's distress. Hersey practiced in Hingham, became a prominent Whig and founded the Hersey professorship at Harvard Medical School, first occupied by Benjamin Waterhouse when the School was founded in 1787.

In all, 26 of the men who attended Harvard College in the classes from 1726 through 1730 practiced medicine. Many were ministers; none had medical degrees. Since some did not graduate or died early, a fair estimate would be that about 5 or 6 per cent of the entire group practiced medicine in one form or another. The need for trained men must have been urgent. Only after John Morgan, William Shippen, Jr., Adam Kuhn and Benjamin Rush, of Philadelphia, with sound training in Edin-

burgh and elsewhere abroad behind them, had set high standards in medical education at their new medical school in 1765 was good medicine available in America. How these men were trained for their task is well told in the biography of *William Shippen, Jr., Pioneer in American Medical Education* (Philadelphia: American Philosophical Society), by Betsy Copping Corner. The gifted author, wife of George Corner, the anatomist, and mother of George, Jr., the obstetrician, not only has written a biographical essay on Shippen but also gives the original texts of Shippen's *Student Diary*, kept in London during 1759–1760. He spent a summer at St. Thomas's, studied anatomy with the Hunters, did not miss seeing Garrick in *Macbeth*, went to an auction of a medical library with John Hunter and heard Mark Akenside give the Harveian oration ("very entertaining") at the Royal College of Physicians.

Readers of detective fiction, as most doctors are, particularly those schooled in the gaslight era of Sherlock Holmes and Dr. Watson, will find in *Bernard Spilsbury, His Life and Cases* (London: George G. Harrap and Company), by Douglas G. Browne and E. V. Tullett, nostalgic moments in reviewing the Crippen case. The trial of that American murderer, with scanty medical qualifications, gave Spilsbury an individual triumph, through his lucid manner in giving the evidence from pathological investigation. At thirty-three he became a national figure, and his name became legendary throughout the world as one of the most distinguished doctors in forensic medicine. He exerted a lasting influence on both pathology and medical jurisprudence, raising the first to a more exact science and the second to the status of an honorable career.

The psychology of the criminal mind offers a fascinating topic for medical evaluation. The study of individual cases offers the best scope for such intellectual activities. Macdonald Critchley, a distinguished neurologist, analyzed the sadistic life of such a criminal in *The Trial of Neville George Clevely Heath* (London: William Hodge and Company), a book in the long and well edited *Notable British Trials Series*. The evidence as produced at the trial is given in full, including that of the physicians, but Critchley's fifty-page introduction holds the most interest. Heath, a sexual psychopath, was hanged, and under the M'Naghten Rules no other course seems justified. Critchley, weighing the doctrine of diminished responsibility, with indefinite sentence, argues against compulsory submission to electric shock, leukotomy and castration. To him "such measures can be regarded as no less repugnant than the scaffold, and indeed in many ways more dangerous."

In the field of bibliography, the medical profession is greatly indebted to John F. Fulton, the Rosenbach Fellow in Bibliography for 1950, for *The Great Medical Bibliographers. A Study in Humanism* (Philadelphia: University of Pennsylvania Press). Following the chronologic line from Tritheim, Champier and Gesner, through the seventeenth and eighteenth centuries with à Beughem, Van der Linden and Haller, he brings one skilfully through the time of Ploucquet, Callisen, Billings, Choulant and Osler to Geoffrey Keynes and the rise of personal bibliography. Although brief, the book is a pioneer in its field, adequate for the space of three lectures. The illustrations are excellent. As might have been expected, the footnotes and appendixes contain valuable material not easily found elsewhere.

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

CONSULTATION CLINICS FOR CRIPPLED CHILDREN IN MASSACHUSETTS

The October schedule of the Consultation Clinics for Crippled Children in Massachusetts is as follows:

Orthopedic Clinic	Date	Clinic Consultant
Haverhill	October 1	William T. Green, M.D.
Greenfield	October 2	Charles L. Sturdevant, M.D.
Lowell	October 3	Albert H. Brewster, M.D.
Salem	October 6	Paul W. Hugenberger, M.D.
Gardner	October 7	Carter R. Rowe, M.D.
Brockton	October 9	George W. Van Gorder, M.D.
Pittsfield	October 15	Edward J. Coughlin, Jr., M.D.
Worcester	October 17	John W. O'Meara, M.D.
Lynn*	October 20	Daniel M. Killoran, M.D., and John J. Crowley, M.D.
Springfield	October 21	Garry deN. Hough, Jr., M.D.
Hyannis	October 23	Paul L. Norton, M.D.
Fall River	October 27	David S. Grice, M.D.

Rheumatic-Fever Clinic	Dates
Fitchburg	October 1, 8, 15, 22, 29
North Reading	October 7, 14, 21, 28

Plastic Clinic	Held	Consultant
Eastern Massachusetts	Semi-monthly	Bradford Cannon, M.D.
Western Massachusetts	Monthly	Joseph M. Baker, M.D.

Physicians referring new patients to clinic should get in touch with the district health officer to make appointments. Patients are seen by appointment only.

*This clinic serves Lynn, Lynnfield, Saugus, Nahant, Revere, Chelsea, Everett and Winthrop. Referrals should be made directly to the Lynn Hospital.

REVISIONS IN COMMUNICABLE-DISEASE REGULATIONS

The isolation and quarantine requirements of the Department were revised on August 12, and the revisions go into effect on October 1. Copies of the revised regulations will be in the hands of all boards of health before the effective date.

One change, the shortening of the period of isolation for poliomyelitis from two weeks to seven days, went into effect on August 12.

Other important revisions included stopping the placarding of diphtheria and scarlet fever; combining

streptococcal (septic) sore throat with scarlet fever; combining Weil's disease with leptospirosis; making Salmonella carriers reportable; and placing typhoid carrier (cholecystitis of typhoid origin) just after typhoid fever in the table.

Further details are as follows:

Anthrax: under isolation of patient add the phrase, "or free of anthrax bacilli."

Diphtheria: restore restrictions on certain adults; no restrictions except for persons living in a household where diphtheria exists and where occupations involve the handling of foods, including milk, or close association with children, who shall be excluded from their occupations until two negative nose and throat cultures taken at intervals of not less than twenty-four hours have been obtained; remove placarding of diphtheria patients.

Lymphocytic choriomeningitis: remove isolation of patient.

Meningococcal meningitis: isolation until three days after beginning of "adequate specific" therapy instead of sulfonamide therapy.

Ophthalmia neonatorum: add as the definition of the condition, "the term of ophthalmia neonatorum includes any acute conjunctivitis, characterized by inflammation, swelling, redness or unnatural discharge from the eye (s) within two weeks of birth."

Poliomyelitis: reduce isolation of patient from two weeks to seven days.

Salmonella carrier: This condition made reportable. Restrictions same as those for typhoid carrier, except food handlers do not receive vaccine.

Scarlet fever: Combine streptococcal (septic) sore throat with scarlet fever. (Isolation of the patient — one week, or two days after the beginning of adequate specific therapy, which must be continued for seven additional days.)

Adult contacts: no restrictions except for food and milk handlers, who may be permitted to work after one week or after two days of specific prophylactic treatment, which must be continued for three additional days.

Child contacts: One week, or two days after the beginning of specific prophylactic treatment, which must, be continued for three additional days.

Remove placarding.

Septic sore throat: omit (combine cases with scarlet fever).

Tularemia: remove isolation of patient.

Typhoid fever: release cultures not to be obtained until ten days after cessation of antibiotic treatment.

Typhoid carrier: change designation from "cholecystitis of typhoid origin" to "typhoid carrier (including cholecystitis of typhoid origin)." (Place after typhoid fever in table.)

Weil's disease: include under "leptospirosis," which will be placed after leprosy in the table.

A new note is added calling attention to the law making it unnecessary for a child to have a certificate to return to school for a disease in which exclusion is not required by board-of-health regulations. (Chapter 89 of the *Acts* of 1952.)

BOOK REVIEW

A Medical History of Persia and the Eastern Caliphate from the Earliest Times Until the Year A.D 1932. By Cyril Elgood, M.A., M.D., F.R.C.P. 8°, cloth, 617 pp. New York: Cambridge University Press, 1951. $10.00.

Dr. Elgood has written the first comprehensive medical history of Persia, now Iran, from the earliest times to the completion of the nationalization of the medical services in 1931. Previously, special study had been made of the great Persian physicians who wrote in Arabic, notably Avicenna and Rhazes, but the later physicians who wrote in Persian had been almost entirely neglected. Dr. Elgood has remedied this condition by discussing fully the works of this last group to about 1794.

The modern period is presented in chapters dealing with the British influence, the East India Company in Persia, the early Qajars (1781–1831), the mission of Sir John McNeill, the introduction of Western medicine, including the history of the Sanitary Council and the Quarantine Service, and the nationalization of the medical services. There is a chapter on early ophthalmology featuring the work of Hunayn, who wrote the earliest known systematic textbook, *Ten Treatises on the Eye*, and of Alhazen and Avicenna. There are also special chapters on Avicenna and Rhazes, two of the great physicians of all time, on Arabian medicine in theory and practice, and on Arabic research and medicine. There is an extensive index. A good, large, easily read type has been used for the printing on a light, nonglare paper. The book is recommended for all history collections, medical and general.

J. F. BALLARD

BOOKS RECEIVED

The receipt of the following books is acknowledged, and this listing must be regarded as a sufficient return for the courtesy of the sender. Books that appear to be of particular interest will be reviewed as space permits. Additional information in regard to all listed books will be gladly furnished on request.

Calculation of Industrial Disabilities of the Extremities. By Carl O. Rice, M.D., M.S., Ph.D., clinical associate professor of surgery, University of Minnesota Medical School, and surgical staff, St. Barnabas Hospital, Minneapolis. With drawings by Jean E. Hirsch. 8°, cloth, 280 pp., with 160 illustrations. Springfield, Illinois: Charles C Thomas, 1952. $10.50.

Dr. Rice has developed a comparatively simple formula for the use of physicians, insurance adjusters and others in the calculation of industrial disabilities. The preliminary chapters deal with the history of the subject, anatomic considerations and relative values. The following chapters discuss in detail fundamental principles, and calculation of disability in the upper and lower extremities. The historical chapter gives an explanation of the methods of McBride, Kessler, Gray and others, and the anatomic illustrates the normal range of motion of the various joints of the extremities. The book should be in all collections on industrial medicine and orthopedics.

Recent Advances in Clinical Pathology. By various authors. General editor, S. C. Dyke, D.M. (Oxon.), F.R.C.P. (Lond.). Second edition. 8°, cloth, 575 pp., with 36 illustrations and 37 plates. Philadelphia: Blakiston Company, 1951. $6.00.

The first edition of this composite book was printed in 1947. The second edition has been thoroughly revised, necessitating the rewriting of practically the whole of the text. Thirty-two specialists have contributed papers, and the material has been divided into four parts: bacteriology; biochemistry; hematology; and histology. There is a special article on laboratory design and equipment. The illustrations are excellent, and there is a good index. The book should be valuable in reference collections on clinical pathology.

NOTICES

AMERICAN ASSOCIATION OF BLOOD BANKS

The fifth annual meeting of the American Association of Blood Banks will be held in Milwaukee, Wisconsin, October 9–11, with headquarters at the Hotel Schroeder.

SPRINGFIELD HOSPITAL EDUCATIONAL PROGRAM

The educational program of the Springfield Hospital for the month of October is as follows:

Wednesday, October 1.

1–2 p.m. Orthopedic Conference in the Staff Conference Room.

Saturday, October 4.

8:30–10 a.m. Surgical Service Conference in the Classroom, second floor.

10–11 a.m. Grand Surgical Rounds in the Staff Conference Room.

11 a.m.–12 noon. Combined Medical-Surgical Grand Rounds in the Staff Conference Room.

Wednesday, October 8.
1–2 p.m. Neurosurgical Conference on "Treatment of Head Injuries" by Dr. Joseph Hahn in the Staff Conference Room.

Saturday, October 11.
8:30–10 a.m. Surgical Service Conference in the Classroom, second floor.
10–11 a.m. Grand Surgical Rounds in the Staff Conference Room.
11 a.m.–12 noon. Clinicopathological Conference in the Staff Conference Room.

Wednesday, October 15.
1–2 p.m. Orthopedic Conference in the Staff Conference Room.

Saturday, October 18.
8:30–10 a.m. Surgical Service Conference in the classroom, second floor.
10–11 a.m. Grand Surgical Rounds in the Staff Conference Room.
11 a.m.–12 noon. Medical Death Review in the Staff Conference Room.

Tuesday, October 21.
8:30 p.m. Guest Speaker Conference in the Staff Conference Room on the subject "Esophageal Diverticulum" by Dr. C. Douglas Sawyer, attending surgeon, Methodist Hospital, Brooklyn, New York.

Wednesday, October 22.
1–2 p.m. Dermatology Conference in the Staff Conference Room by Dr. Leonard E. Anderson.

Saturday, October 25.
8:30–10 a.m. Surgical Service Conference in the Classroom, second floor.
10–11 a.m. Grand Surgical Rounds in the Staff Conference Room.
11 a.m.–12 noon. Surgical Death Review in the Staff Conference Room.

Wednesday, October 29.
1–2 p.m. Pediatric Conference in the Staff Conference Room presented by Dr. H. H. Shuman.

Every Wednesday from 11 a.m. to 12 noon a Psychosomatic and Group Psychotherapy Conference is held in the Staff Room under the direction of Dr. Calvert Stein.

Every Thursday at 3 p.m. a Pathology Conference is held in the Department of Pathology.

Every Friday from 1 to 2 p.m. a Radiology Conference is held in the Department of Radiology.

SOCIETY MEETINGS AND CONFERENCES

SEPTEMBER 26–28. Aero Medical Association. Page ix, issue of July 31.

OCTOBER 1. Essex North District Medical Society. Page 226, issue of August 7.

OCTOBER 1–3. Mississippi Valley Medical Society. Seventeenth Annual Meeting. Jefferson Hotel, St. Louis, Missouri. Page xiii, issue of August 21.

OCTOBER 1–3. American Medical Writers' Association. Page ix, issue of July 31.

OCTOBER 1–29. Springfield Hospital Educational Program. Page 497.

OCTOBER 1–APRIL 8. Postgraduate Medical Institute. Page 452, issue of September 18.

OCTOBER 3, NOVEMBER 18, FEBRUARY 6 AND APRIL 3. New England Society of Anesthesiologists. Page 418, issue of September 11.

OCTOBER 6–17. New York Academy of Medicine. Page xi, issue of July 24.

OCTOBER 8. New England Pediatric Society. Page 338, issue of August 28.

OCTOBER 9–11. American Association of Blood Banks. Page 497.

OCTOBER 14. Springfield Academy of Medicine. Page 190, issue of July 31.

OCTOBER 18–23. American Academy of Pediatrics. Page xi, issue of July 24.

OCTOBER 27–29. Medicomilitary Symposium. Page 454, issue of September 18.

OCTOBER 28–30. New England Postgraduate Assembly. Hotel Statler, Boston.

OCTOBER 29. New England Obstetrical and Gynecological Society. Annual Meeting. Hotel Somerset, Boston.

OCTOBER 30–NOVEMBER 1. National Association for Music Therapy. Page ix, issue of July 31.

OCTOBER 31 AND NOVEMBER 1. Eastern Section of American Trudeau Society. Page 190, issue of July 31.

NOVEMBER 2–7 AND 10–21. Courses in Laboratory Diagnosis of Tuberculosis. Page 354, issue of February 28.

NOVEMBER 9 AND 10. American Society for the Study of Arteriosclerosis. Page 78, issue of July 10.

NOVEMBER 11–14. American Society of Anesthesiologists, Inc. Third annual refresher course program. The Bellevue-Stratford, Philadelphia.

DECEMBER 7. American Academy of Dental Medicine. Hotel Statler, New York City. Page 338, issue of August 28.

JANUARY 5, FEBRUARY 2, AND MARCH 2, 1953. Oak Ridge Institute of Nuclear Studies. Page 454, issue of September 18.

JULY, 1953. International Congress on Medical Librarianship. Page 316, issue of February 21.

DISTRICT MEDICAL SOCIETIES

ESSEX SOUTH
OCTOBER 1. 6:45 p.m. Amesbury Country Club, Amesbury.

MIDDLESEX SOUTH
OCTOBER 29. 11:15 a.m. Mount Auburn Hospital, Cambridge.

NORFOLK
OCTOBER 28.
NOVEMBER 25.
JANUARY 27.
FEBRUARY 24.
MARCH 24.

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING THURSDAY, OCTOBER 2

THURSDAY, OCTOBER 2
*8:30–10:00 a.m. Orthopedic Staff Conference, Boston City Hospital.
*9:00–10:00 a.m. Cardiac Grand Rounds. Main Amphitheater, Children's Medical Center.
*9:00–10:00 a.m. Arthritis Grand Rounds. Robert Breck Brigham Hospital.
*11:00 a.m.–12:30 p.m. Hand Clinic (Physical Medicine and Rehabilitation Service). Boston City Hospital.
*5:00–6:00 p.m. Surgical Division Lecture. Mount Auburn Hospital, Cambridge.

FRIDAY, OCTOBER 3
*10:00 a.m. Tuberculosis Surgical Clinic. South End Health Unit, 57 East Concord Street.
*10:00 a.m.–1:00 p.m. Medical and Surgical Grand Rounds. Dr. George W. Thorn and Dr. Francis D. Moore. Main Amphitheater, Peter Bent Brigham Hospital.
10:30–11:15 a.m. Diabetes Lecture. Dr. Elliott Joslin. New England Deaconess Hospital.
*11:00 a.m.–12:00 m. Pediatric Conference. Mount Auburn Hospital, Cambridge.
*1:30 p.m. Fertility and Endocrine Clinic. Free Hospital for Women, Brookline.
7:00–9:00 p.m. Combined Anesthesia Teaching Conferences of Boston. Amphitheater, Evans Memorial, Massachusetts Memorial Hospitals, Eighth Floor, 65 East Newton Street, Boston.

SATURDAY, OCTOBER 4
*9:00 a.m. Proctology Clinic. Peter Bent Brigham Hospital.
*9:00 a.m. Alcoholism Clinic. Peter Bent Brigham Hospital.
*11:00 a.m.–12:00 m. Blood Clinic. Dr. William Dameshek. Stearns Auditorium, New England Center Hospital.

MONDAY, OCTOBER 6
*8:00–9:00 a.m. Combined Medical and Surgical Rounds for Diabetic Surgery Cases. New England Deaconess Hospital.
*8:30–9:30 a.m. Clinic by entire Surgical Staff and Anesthesia Staff. Cheever Amphitheater, Dowling Building, Boston City Hospital.
*12:00 m. Clinicopathological Conference (Boston Floating Hospital). Pratt Lecture Hall, Joseph H. Pratt Diagnostic Hospital.
*12:15–1:15 p.m. Clinicopathological Conference. Main Amphitheater, Peter Bent Brigham Hospital.
*7:00–9:00 p.m. Anesthesia Teaching Conference. Stearns Auditorium, New England Center Hospital.

TUESDAY, OCTOBER 7
*9:00 a.m. Geriatrics Clinic. Peter Bent Brigham Hospital.
*12:00 m.–1:00 p.m. Pediatric Rounds. Burnham Memorial Hospital for Children, Massachusetts General Hospital.
*12:15–1:15 p.m. X-Ray Conference. Dr. Merrill Sosman. Main Amphitheater, Peter Bent Brigham Hospital.
*5:00–6:00 p.m. Service Meeting followed by Clinical Conference (Medical, Surgical, Obstetrical). Faulkner Hospital.

WEDNESDAY, OCTOBER 8
*12:00 m. Boston State Hospital Psychiatric Seminar. Reception Building Auditorium, 591 Morton Street, Dorchester.
*12:00 m.–1:00 p.m. Medical Conference. (Children's Medical Center.) Jimmy Fund Building, 25 Binney Street.
12:30 p.m. Weekly Pathological Meeting. Joslin Amphitheater, New England Deaconess Hospital.
*4:00 p.m. Thoracic Conference. Held by Overholt Clinic, Harris Hall, New England Deaconess Hospital.
*4:00–5:30 p.m. Fracture Lecture. Boston City Hospital.

*Open to the medical profession.